DROSOPHILA INFORMATION SERVICE
Number 35

July 1961

(Issued in 1,000 copies)

TABLE OF CONTENTS

D. melanogaster
Stock Lists
United States
Ames, Iowa 34: 9
Amherst, Massachusetts 34:10
Austin, Texas 35: 6
Baltimore, Maryland 35: 7
Berkeley, California 35: 7
Buffalo, New York 35: 8
Chapel Hill, North Carolina 35: 9
Chicago, Illinois 35: 9
Cleveland, Ohio 35:10
Cold Spring Harbor, New York 34:11
DeKalb, Illinois 34:12
East Lansing, Michigan 34:12
Gainesville, Florida 34:13
Lafayette, Indiana 35:11
Lawrence, Kansas 35:11
Lexington, Kentucky 35:11
Los Angeles, California 34:14
Minneapolis, Minnesota 35:11
New Haven, Connecticut 34:14
New York, New York 34:26
Norman, Oklahoma 34:15
Oak Ridge, Tennessee 35:11
Pasadena, California 34:16
Philadelphia, Pennsylvania 35:17
Salt Lake City, Utah, Dept. of Surgery 34:26
Salt Lake City, Utah, Dept. of Genetics 35:32
Syracuse, New York 35:32
Tucson, Arizona 35:32
University Park, Pennsylvania 34:27
Urbana, Illinois 34:27
Argentina 35:33
Australia
Adelaide, South Australia 34:28
Brisbane 35:34
Melbourne, Victoria 34:28
Sydney, New South Wales 34:29
Austria 35:34
Belgium 34:29
Brazil 35:34
Canada 35:35
Chile 34:30
Denmark 34:30
Finland 34:30
France 35:35
Germany
Berlin-Buch and Berlin-Dahlem 34:32
Gottingen 35:35
Hamburg 35:36
Hamburg-Eppendorf 35:36
Heidelberg 34:33
Karlsruhe 34:33
Marburg/Lahn 34:33
Mariensee 34:34
Tübingen 34:35
Ghana 34:35
Great Britain
Bayfordbury 35:36
Birmingham, England 34:35
Edinburgh, Scotland 34:36

Glasgow, Scotland 35:36
Harwell, Berks, England 35:37
London, England 34:36
Manchester, England 34:37
Sheffield, England. 34:37
India
Calcutta, Indian Statistical Institute. 34:37
Calcutta, University of Calcutta 35:38
Israel 34:38
Italy
Milano. 34:38
Naples 34:39
Pavia 34:39
Japan
Anzyo, Aichi. 34:40
Chiba-Shi 34:40
Hiroshima 35:38
Kyoto 34:41
Misima. 34:42
Mitaka, Tokyo 35:38
Osaka 34:43
Sapporo 34:44
Tokyo 34:44
Korea
Kongju, Chung Cheong Do 34:45
Kwangju 34:45
Seoul 34:46
Netherlands
Leiden, Rijksuniversiteit, Laboratorium Voor Stralengenetick. . . . 35:38
Leiden, Rijksuniversiteit, Genetisch Laboratorium 35:39
Utrecht 35:39
Norway 34:46
Spain 35:41
South Africa 35:40
Sweden
Stockholm 35:42
Uppsala 35:43
United Arab Republic 34:47
New Mutants
Report of A. B. Burdick. 35:45
Report of W. W. Doane. 35:45
Report of W. W. Doane. 35:45
Report of J. L. Hubby. 35:46
Report of P. T. Ives 35:46
Report of James Kidwell. 35:46
Report of H. W. Lewis. 35:46
Report of A. Schalet 35:46
Report of A. H. Sturtevant 35:47
Report of V. Tinderholt. 35:47
Linkage Data
Nash, D. J. and E. C. Keller, Jr. 35:47
Other Drosophila Species
Stock Lists
United States
Pittsburgh, Pennsylvania. 35:48
Rochester, New York 35:48
France 35:49
Italy. 35:49
Korea. 35:50
Spain. 35:50
Bibliography 35:51
Notes and News
Research Notes
Angus, D. Drosophila collection from the Territory of Papua-New Guinea 35:71

Barbour, Evelyn and S. Zimmering. Preliminary analysis of a Y Chromosome from nature carrying a mutant allele of bobbed 35:71
Bateman, Angus J. X-ray induced "crossing-over" 35:71
Beatty, R. A. and N. S. Sidhu. A note on the occurrence of bulbous testes ends in Crianlarich strain of D. melanogaster. 35:72
Brosseau, George E., Jr. The effect of M(2)S10 on the fertility of some compound XY chromosomes 35:73
Burdette, W. S. Effect of penicillin on mutation rate following irradiation in different concentrations of oxygen 35:73
Burdick, A. B. 1(2)55i at Erie, Pennsylvania. 35:75
Carlson, E. A. and R. Sederoff. A selective scheme for recovering pseudoallelic recombinants, "conversion" phenomena, and reverse mutations 35:75
Chandley, A. C. Mutations induced in presumed spermatocytes 35:76
Chandley, A. C. Timing spermatogenesis in D. melanogaster with tritiated thymidine 35:77
Divelbiss, J. E. A sterility factor affecting both males and females in D. melanogaster. 35:77
Doane, W. W. Persistence of fs(2)adp in the Kaduna population after four years 35:78
Dorn, G. L. and A. B. Burdick. Recombination between Df(1)259-4 and various mutants of the miniature-dusky complex in D. melanogaster 35:78
Ehrman, Lee. Mutant genes in the Transitional subspecies of D. paulistorum 35:79
Fabergé, A. C. and B. H. Judd. Chromosome breaks by alpha particles . 35:79
Fox, Allen S. and Eileen A. Sweeney. Chemical structure and time of appearance of the sex peptide of males in D. melanogaster 35:81
Frost, J. N. Double fertilization mosaics 35:81
Frost, J. N. Two mosaics of unusual origin 35:81
Frye, Sara. Evidence that achaete may not be to the right of yellow . 35:82
Frye, Sara. Frequency of "transmissible mutation" at the w and f locus in the scute-8 chromosome in relation to X-ray dose in Drosophila 35:82
Frye, Sara. Persistence of qualitatively diverse "yellow" mutants in scute-8 chromosomes in the absence of selection for one year . . 35:83
Frye, Sara H. Spontaneous "yellows" as gross rearrangements in Drosophila. 35:83
Fuscaldo, Kathryn E. and Allen S. Fox. Immunogenetic studies of white-variegated position effects 35:84
Ghini, Clara. Effects of nebularine and EOC (8-etoxy caffine) on selection response for sternopleural hairs in D. melanogaster . . . 35:84
Grell, R. F. The penetrance of sparkling-Cataract 35:85
Hildreth, P. E. and J. C. Lucchesi. Fertilization in D. melanogaster and D. virilis 35:85
Hochman, B. On the viability of the brown-Variegated^{1}/brown-Variegated^{57e} heterozygote 35:85
Hunter, Alice S. and Sara Newball. Drosophila of Old Providence Island 35:86
Imaizumi, T. XXY strain derived from the wild Miyazu stock of D. melanogaster and its lethality 35:87
Ives, P. T. More data from ras^{2}/ras^{4} and y/y^{2} recombination tests . . 35:88
Jacobs, M. E. Influence of ebony and + alleles on oxygen consumption and egg production in D. melanogaster 35:89
Kato, M. Influences of essential fatty acids on the growth and egg productivity of D. melanogaster 35:89
Khishin, Aziz F. Induction of mutations in D. melanogaster by "immersion" in solutions 35:89
Mead, Charles G., and Allen S. Fox. The characterization of the deoxyribonucleic acids of D. melanogaster 35:89

Meyer, Helen U. and Michael L. Criswell. Crossover analysis of sex-linked mutations induced in oogonial cells by repeated treatments with 4000r of X-ray 35:90
Meyer, Helen U. and Evelyn R. Meyer. Sperm utilization from successive copulations in females of D. melanogaster 35:90
Novitski, E. Post-treatment of irradiated sperm by low temperature . 35:92
Parker, D. An apparent incompatibility among seemingly normal members of the species D. simulans 35:92
Roberts, Paul A. Bristle differentiation in genetic mosaics of D. melanogaster 35:92
Sandler, L. and C. W. Cotterman. A possible interpretation of the conversion of X chromosome by SD 35:93
Sederoff, R. and E. A. Carlson. The relation between allelic phenotype and allelic localization within the dumpy region 35:93
Seto, F. The relative constancy of phase specific action of recessive lethal factors in D. melanogaster 35:94
Spiess, E. B. and R. B. Helling. Linkage of chromosome II lethals in D. melanogaster 35:95
Stern, C. and E. Sherwood. A search for maternally influenced sex-ratio in D. melanogaster 35:96
Strangio, V. A. Radiosensitivity to certain breakage aberrations during spermatogenesis in D. melanogaster 35:96
Tates, A. D. and F. H. Sobels. The genetic effects of post-radiation treatment with cyanide in pupal spermatids. 35:98
Terzaghi, Eric and E. Novitski. An attempt to produce fertile "transformed" males 35:99
Tokunaga, Chiyoko. Notes on the sex chromosome constitution of oogonial cells in gynanders 35:100
Wolff, M. and A. Coughlin. Tests for meiotic drive in interspecific hybrids 35:100
Würgler, Friedrich E. Modification of X-ray induced embryonic mortality by different anoxia conditions before and during irradiation of uncleaved D. melanogaster eggs 35:102
Zimmering, S. and H. J. Muller. Studies on the action of the dominant female-lethal Fl and of a seemingly less extreme allele, Fl^{s} . . . 35:103

Technical Notes

Browning, Luolin S. and Edgar Altenburg. Weighting of dehydrated Drosophila as a counting method 35:105
Mickey, G. H. Nigrosine as an aid for staining brain and salivary gland chromosomes 35:106
Moyer, S. E., R. E. Comstock, and L. H. Baker. Efficient procedures for culturing Drosophila in disposable paper containers 35:106

Personal and Laboratory News 35:107
Materials Requested or Available 35:107
Announcements 35:107

AUSTIN, TEXAS: UNIVERSITY OF TEXAS
Department of Zoology, Genetics Foundation

Wild Stocks

1 Austin, Texas
2 Canton-S
3 Espanola, New Mexico
4 Oregon-R

Chromosome 1

amx/In(1)dl-49, m^2 g^4
amx lz^g v/y f:=
Bx^3 ma-l
l(1)7/dl-49, y Hw m^2 g^4
l(1)7e, l(1)7/dl-49, y Hw
l(1)55a B/In(1)sc^{S1}, S,
w^a/B/sc^8
lz^3/y f:=
m
ma-l
pn^2
Qd
sc cho
sc ec cv ct^6 v g f/FM3,
y^{31d} sc^8 dm B l
sn^3 lz^g v/y f:=
sn^3 m ma-l
su-s sn^2
v^1
v^{58k}
w m
w_a sn^3
w^a rb
w_{a2} spl ec
w_{bf} f^5
w_{bf2} f^5
w_{bl}
w_{bl}
w_{Bwx} spl sn^3/y f:=
w_{Bwx} w^a spl sn^3/y f:=
w_{cf}
w^e
w_h spl sn^3
w^h sn^3 m_N
w_i f^3 bb
w^{sat}
y car/y f:=
y ct^6 ras^2 f
y sc
y sc m_{f^5}
y sc w^{Bwx}/y f:=
y sc z w^a spl ec/y f:=
y W ec f
y w_e spl sn^3
y_2 w^i
y_2 sc w^i
y_2 sc w^a ec
y_2 su-w^a w^a
y_2 su-w^a w^a spl sn^3
y^2 su-w^a z sp^3 w
(sp^3-w=sp-w^{59a29})
y^2 w^a
z
z w^{11E4}
z w^a/y f:=
z w^{Bwx} spl

Chromosome 2

al dp b pr blt bw/
Cy, al^2 lt^3 L^4 sp^2
al dp b pr c px sp/Cy
al S ast ho/Cy, E^{-S}
al S ho/Pm ds^{33k}
ast^4 dp cl
b
b pr c px sp
b vg
Bla/SM5, al^2 lt^v Cy sp^2
Ba/In (2LR), dp
Bl L^2/SM5
bri
bs^2
bw
bw ba
cn bw
ds dp
ds S G $b_2$pr/
Cy al^2 lt^3 L^4 sp^2
ex
ft
G^{rv}/Cy dp^2
ho ed cl
ho ed dp cl
pi/SM5, al^2 lt^v Cy sp^2
pr_2
Pu^2/SM1, al^2 Cy sp^2
S^{56F}, Cy Pm/Gla
Sp J L^2 Pin/Gla

Chromosome 3

cu kar
Gl Sb/LVM
H Pr/In(3R)C, e
ma
ma fl_2
ma ry^2/TM1 Me
mo/Ubx
on ry^2
ri bod e^s/Me, In(3R)C,
Sb e l(3)e
ri p^p
R Ly/In(3L)P, gm^{G130}
R Sb Bd^{32y}/Ubx130
R Sb H^{32y}/Ubx130
ry^2
Sb H/In(3R)c, cd^{101}
Sb/In(3LR), Ubx
st
st c3G ca/TM1, Me ri(sp^2)
tra/In(3LR), Ubx130
(FMA3/w^a v)
Wine/C(3)X
W Sb/In(3LR)ex

Chromosome 4

ci^D/ey^D
pol

Attached X-Y

v f_LB X-Y/y^2 su-w^a w^a bb
X·Y^L y_Sw(-8d)/y f/Y"
X Y^L·Y^S(108-9 Parker)y^2 su-w^a w^a
(bb$^+$)Y^L·Y^S/y $y_L$bb/0
X·Y^S y w/y v f/Y^{L2}
y_S su-w^a w^a bb/0/v f B XY; pol
Y^SX·Y^L, In(1)EN -49, y v f car/
y^2 w^a bb/0

Inversions

In(1)FM6, FM6, y^{31d} sc^8 B dm / y w^{m4} L·$N^{264-84R}$ sn/Y dm sn

y Mul-5, In(1)sc^{S1},S, y w^a B sc^8
y v Muller 5, In(1)sc^{S1},S, y w^a v B sc^8
In(1)y^4, lz^{y4}/y v car

Modified Y

YB^S
y/y/sc^8_S·Y
Y^L/f_L·Y^S/sc v f:=
sc·Y_2^L/In(1)dl-49, y v f·Y^S
Y^S_2/g^2 B·Y^L/y_Lf:=
Y^S/$y_S$$ct^6$ f·Y^L/y f:=
Y^S·Y^S # 2/y v f·Y^L/y f:=

Multichromosomal

sc br w^a R m f; bw(Austin, Texas)(1;2)
v^1; bw(1;2)
w^{col}; bw(1;2)
y^2 w^a spl ec; SM1, al Cy sp^2/In(2LR)102 ds^w sp^2; In(3LP, 3RC)Sb e^s/Ubx^{130} e^s(1;2;3)
y f:=; al; st; pol(1;2;3;4)
ma-1; st(1;3)
w^a; st(1;3)
w^{Bwx}; st(1;3)
w^e; st(1;3)
y v car/Y; pol(1;4)
cn; ma(2;3)
Cy/S; D/C III z(2;3)
dp^v; vo^3(2;3)
pr; st(2;3)
SM1, al Cy sp^2/In(2LR)102 ds^w sp^2; In(3LP, 3RC)Sb e^s/Ubx^{130} e^s(2;3)
SM1/dp b Pm; C Sb/Ubx^{130} e^s(2;3)
SM1/dp b Pm; Ubx^{130}/C Sb (2;3)

Deficiencies

y w^{258-11}/y Hw dl-49 m^2 g^4

Duplications

y w f/y w^{def} rst^3; Dp w^{+51b7}
w^{def} rst^3 car/y w_vf:=; Dp w^{+51C20}
y w^{258-45}; Dp(3)w^v, co/y f:=

Translocations

T(1;4)B^S/Y f:=; bw; e; ci ey^R
T(1;4)N^{264-12}/In(1) dm^5/ci D^{-49}, w lz^s
T(1;4)w^{258-18}/ey^D
T(1;4)w^{258-21}, y w/Ins(1) w^a lz B D^{-49}, sc^8,y^{31d}
T(Y;2)C/pr cn
T(Y;2)G/b H bw
T(2;3)Xa/1(3)Xa_DR
T(3;4)c, Ubx/ci^D

Triploid

$\frac{y^2\ sc^8 w^a\ ec}{sc^8 \cdot Y,\ y}$/FM4, y^{31d} sc^8 dm B; cn/cn/cn;
v f/y Mul-5

BALTIMORE, MARYLAND: THE JOHNS HOPKINS UNIVERSITY
Department of Biology

Note: Additions and corrections to the list in DIS-33 (p. 12).

Additions:

c9 w m f
c9a w_2m f (containing XXY ♀♀)
c9b y^2 cho^2
c31 In(1)X^{c2} w^{vc}/y w lz^s ♀♀ & y w lz^s/sc^8·Y ♂♂
d7a cn Su-Pm/Cy cn vg Pm
d7b cn Su-Pm Tac/Pm (dp b c ?)
d9a l(2)mg
d10a mi/Pm^2
d11c px slt sp
e3a l(3)tr/Mé Sb
e6a red
g1e cn_4 bw; e
g1f ct^{45e} v; bw; e; $(ey^2)^+$
g3c Cy/tu bw; st su-tu
g9a v; bw; e
g9b y2 v f; bw
g11a T(Y;2) J/px bw sp

Discarded:

c24 car l^{B3+1}/Ins(1)sc^{S1} sc^8, B w^a
c25 car l^{B5+2}/Ins(1)sc^{S1} sc^8, B w^a

BERKELEY, CALIFORNIA: UNIVERSITY OF CALIFORNIA
Department of Zoology

Wild Stocks

1 Canton-S
3 Samarkand-inbred
5 $+^3$

Chromosome 1

100 B
102 br
103 br ec/y^{3d}
104 Bx
105 Hw^{49c}/FM1, y^{31d} sc^8 w^a lz^s B
106 In(1)dl-49, y Hw m^2 g^4/y f:=
107 kz g^2 B/y
108 $Df(1)N^8$/In(1) dl-49, m^2 g^4
109 sd
110 sx vb^2 sy/In(1)AM
112 v car
114 w
115 w^{bl}
116 w^{bl}/FM_4
117 w^{ch} wy
118 w^{ch} wy/FM_4
119 w^{co} sn^2
120 w^{co} sn^2/FM_4
121 w^e bb^1/ClB (with extra Ys)
122 w^{vc}
123 y
124 y ac/y
125 y ac sn^3
126 y ac v
127 y ac w^a
128 y ac Dp $w^a(w^a)_2$/y^2 ac w^{561} ec
129 y ac Dp $w^a(w^a)_2$/ y f:=
130 y sc
131 y sc m f^5
133 y w
134 In(1)y,In(1)w
135 y w spl sn^3/y f:=
140 y^2 cv v f
141 y^2 sc w^a ec/y ac w^{bl}
150 Muller-5; y sc^8·Y
160 X^{c2} f car/y f:=

Chromosome 2

200 a px sp

201 al b c sp^2/In(2LR)Cy, al^2 lt^3 L^4 sp^2
202 al b pr cn vg c sp^2/In(2LR)Cy, L^4 sp^2
204 al dp b pr blt bw/Cy, al^2 lt^3 L^4 sp^2
205 al dp b pr c px sp/Cy pr
206 al dp b pr cn vg c a px bw mr sp/S^2 Cy lt^3 pr^+ Bl cn^2 L^4 sp^2
208 b
212 bw
214 c
215 cg-c/U
216 cl
218 cn bw
220 esc c sp/SM5, al^2 Cy lt^v sp^2
225 l(2) gl cn bw/Cy, al^2 lt^3 L^4 sp^2
226 L^4
228 pr cn ix/SM5, al^2 lt^v Cy sp^2
229 pr en
232 vg
233 vg^{no}

Chromosome 3

301 cu
303 cv-c sbd^2
308 Gl Sb/LVM
310 h
312 Ly/D^3
314 p^p
315 ru h st p^p ss e^s
316 ru h th st cu sr e^s ca
319 se
320 se h
323 ss
324 ss^a
325 ss^a-B
340 In(3LR)TM1, Mé/In(3LR)Rbx^{130} e^s
350 Pc/T(2,3)Mé

Chromosome 4

402 bt ey^R sv^n
403 bt^D/ci^D
404 ci
405 ci^W
408 ci ey^R 412 ey^2
420 M-4/ey^D

Multichromosomal

510 w; vg
511 X^{c2} t/y f:=; en
512 y ac sn^3; stw^3 en
513 y ac sn^3/Muller-5; en
516 y f:=; bw; e; ci ey^R
517 w^av/y v; tra/C(3)x
520 b; p^p
521 Cy/Pm; D/Sb
522 vg; se
530 se h; ci ey^R

Translocations

603 T(1;2)Bld/ClB
606 T(1;2)sc^{19}/y f:=; fes sc^{19i} b pr/Cy, dp^{Th} pr
607 T(2;3)Xa/Sb Ubx

Multiple inversions (Weaver)

A sn^3; $\frac{Cy}{cn\ bw}$;ri (Pasadena)
B y; $\frac{Cy}{cn\ bw}$;ri (Pasadena)
C sn^3; $\frac{Cy}{cn\ bw}$;ri (Berkeley)
D y; $\frac{Cy}{cn\ bw}$;ri (Berkeley)
E sn^3; $\frac{Mé}{bw}$;ri
F y; cn bw; $\frac{Ubx}{ri}$
G sn^3; cn bw; $\frac{Ubx}{ri}$
H y: $\frac{Pm}{cn\ bw}$; ri (Pasadena)
I sn^3; $\frac{Pm}{cn\ bw}$;ri (Berkeley)
J y; $\frac{Gla}{cn}$;ri
K sn^3; $\frac{Gla}{cn\ bw^D}$;ri
L y; $\frac{In\ bw^D}{cn\ bw}$;ri
M sn^3; $\frac{In(2L)Cy}{cn}$ bw;ri

BUFFALO, NEW YORK: STATE UNIVERSITY
College of Education
(George M. Lang, Assoc. Prof. of Science)

Wild Stocks

Sex Linked

eosin eye
white eye

Chromosome 2

black body
brown eye
curved wing
vestigal wing

Chromosome 3

scarlet eye

Chromosome 4

eyeless

CHAPEL HILL, NORTH CAROLINA: UNIVERSITY OF NORTH CAROLINA

Wild Stocks

1 Oregon-R: isogenics, mixed

Chromosome 1

2 f^5 su-f
3 v f BB
4 v f BBB/y v f car
5 v f ++: reverted from v f BB
6 w^{57}
7 w^e sn/ClB
8 y^{58}
9 y v f car/y w

Chromosome 2

10 al
11 al b lt c sp
12 al b pr stw c
13 al b pr stw c px
14 al b pr stw c sp
15 al b pr stw px
16 al b pr stw sp
17 al Cy L sp/al dp b pr lt stw c px sp
18 al Cy lt L sp/al dp c px sp
19 al Cy pr stw c sp/al dp b pr lt stw c px sp
20 al Cy pr stw px/al sp pr lt stw c px sp
21 al Cy stw L sp/al dp pr stw c sp
22 al sp Cy L sp/dp lt l sp
23 al lt stw sp
24 al sp
25 b pr lt stw
26 b pr stw c
27 b pr stw c sp
28 dp b lt sp
29 sp b pr str px sp
30 dp lt c px
31 dp lt c sp
32 dp lt px
33 dp lt sp
34 dp Pm/Cy lt L^4
35 dp pr stw px
36 Pm/Cy pr stw L
37 S L^4/Cy lt Pfd
38 S^W Cy pr/Pfd L^2
39 S Pfd/Cy lt cn^2 L^4
40 sp
41 Sp Bl L^2/lgl Cy cn^2
42 Sp Bl L bw^D/Cy cn^2
43 vg: isogenic in 1959

Chromosome 3

44 M(3)y Gl/Sb Ubx
45 M(3)y Gl Inv(3R)LVM/ Inv(3L)LVM Sb Ubx
46 M(3)y Gl p^p cu/p^p cu Sb Ubx
47 M(3)y Gl Sb Ubx/LVM
48 M(3)y Sb/Gl Ubx
49 M(3)y Ubx/Gl Sb
50 Me cu sr e^s ca/ru h th st cu sr e^s ca ("rucuca")
51 ru h th st cu su e^s ca ("rucuca")
52 ru h th st sr e^s Pr ca

Multichromosomal

53 ClB/w^e sn; Sp Bl L^{rm}/Cy
54 f BB; th st cu sr
55 b pr stw sp; th st cu sr
56 Cy/Pm; H/Sb C sr
57 Sifter: Muller
58 Sp Bl L/T (2:3) Mé

CHICAGO, ILLINOIS: UNIVERSITY OF CHICAGO
Department of Zoology

Note: Only stocks not commonly carried in other laboratories are listed.

Wild-type

1 Chicago wild-type

Chromosome 1

2 fu^{57a}/FM-1
3 l(1)J-1, sc^{J-1}/Del (1)24
4 lix/y w
5 sc^2 pn
6 sc^{10-1}/y Hw
7 sc z^m
8 $w^e bb^1$/y f:=
9 y ac z ec ct
10 y z^Q

Chromosome 2

11 b pr lt stw^3
12 bw^D
13 bw^{59}
14 bw^{75}
15 bw^{81}
16 bw^5-/Cy cn^2 L^4 sp^2
17 In(2R)bw^{v34}, Cy/al dp b Bl c px sp
18 In(2LR)lt^{m3}
19 T(2;3)lt^{m29}
20 pr ltd

Chromosome 3

21 C_3G
22 ru h th st cu sr e^s ca

Chromosome 4

23 spa^{Cat}/ci^D

Inversions-X

24 In(1)EN, y/y f:=
25 Ins(1)sc^4EN, y sc^4 car y/y f:=
26 Ins(1)$sc^{S1}$$sc^8$, y sc^{S1} car n w^a sc^8/In(1)dl-49, y w lz^s
27 In(1)y^4, y^4

Deficiencies - Duplications - X Chromosome

28 w^{-5gK13} spl; Dp(1;3)w^{vco}
29 Df(1)w^{258-45}, y w^- spl dm; Dp(1;3)w^{vco}/y w f

30 Df(1)w^{258-48}, y w^-; Dp(1;3)w^{vco}/y w f
31 y w^- rst^3; Dp(1;2R)w^{51b7}
32 Dp(1)w^{vc} 6094b/Y^S wy·$Y^L$$y^{55f10}$/y w
33 Dp(1;3)$w^{m264-58a}$/+; y w f Y^L·Y^S/
y w/sc^8·Y

Ring-X

34 X^{c1}, y/sc^8·Y/y v f car
35 X^{c2}, y cv v f car

Reversed Ring

36 RR, In(1)EN, car f v y /In(1)sc^8,
y^- ac^- sc^- m/sc^8·Y

Tandem Metacentric

37 TM(Hw f), originally y Hw v f·y^+
cv f y^+

Reversed Acrocentric

38 RA, y ac sc pn -- In(1)sc^8/In(1)sc^8
(C.O.J-3), y^- ac^- sc^8 w^a f/sc^8·Y,y

X with Y Fragments Attached

39 FR1, Y^s y cv v f/y f:=
40 y^2 su-w^a w^a Y^L·Y^L B^S/Ins(1)sc^{S1}
dl-49, v
41 y w^a Dp B^S/Ins(1)sc^{S1} dl-49, v
42 y w f Y^L·Y^S/sc^8·Y/y w
43 y Hw·Y^S y^+/Y^{cL}/y w^a:=

Attached X-Y; no free Y

44 Y^S B f v y·Y^L y^+/0/y v bb
45 Y^S y B·Y^Ly/y v f/0 with sc^{J4}
46 Y^S w y·Y^L y^+/0/y v bb
47 y^2 su-w^a w^a Y^L·Y^S/y bb/0
48 y w f Y^L·Y^S/0/y w

Altered Complete Y's

49 Y:bw^+/y v; bw
50 sc^8·Y:bw^+/y v f/y f:=; bw
51 sc^8·Y, y^{54e} ac^{54e}/y v; bw

Y^S Fragments

52 Y^{cS};bw^+ bb^+/g^2 B·Y^L/y v bb; bw
53 Y^S/g^2 B·Y^L/y f:=
54 Y^S:y^+ bb^+-5/B·Y^L/y w
55 Y^S:y^+ bb^+-6/g^2 B·Y^L/y v bb; bw
56 Y^S:y^+ bb^+-7/g^2 B·Y^L/y w
57 Y^S·Y^S/sc sc^8·Y^L/y^2 su-w^a w^a bb
58 Y^S·Y^S #2/y v f·Y^L/y f:=
59 sc^{V1}·Y^S/y v f bb·Y^L/y f:=

Y^L Fragments

60 sc·y^L/y ac w^a ct^6 f·Y^S/y f:=
61 sc^{S1}·Y^L #2/y ct^6 f·Y^S/y w
62 Y^{cL}/y ct^6 f·Y^S/y w^a:=
63 Y^L-13/y ct^6 f·Y^S/y v bb; bw
64 Y^{cL}-14/y ct^6 f·Y^S/y v bb; bw
65 Y^{cL}-15/y ct^6 f·Y^S/y v bb; bw

Multichromosomal

66 In(3LR)Ubx^{130}, Ubx^{130} e^s/Xa
67 SM1, al Cy sp^2/In(2LR)102 ds^w sp^2;
In(3LP,3RC)Sb e^s/Ubx^{130} e^s
68 y/sc^8·Y; ru h th st p^p cu sr e^s
69 w; Cy/Pm; CxD/Sb, In(3R)Mo
70 y; In53A, al^2 Cy sp^2; Ubx^{130} e^s/Xa
71 y w/Y^S f y·Y^L y^+; sv^n
72 Y^S:$y^+$$bb^+$-7/$g^2$ B·Y^L/±;Cy/Pm;CxD/Sb,
In(3R)Mo
73 Y^S:y^+ bb^+-5/±/B·Y^L;Cy/Pm;CxD/Sb,
In(3R)Mo

CLEVELAND, OHIO: WESTERN RESERVE UNIVERSITY

Chromosome 1

1 ec dx
2 sc cv v f
3 v
4 w
5 y sc v g x y f:=
6 y w sn^3

Chromosome 2

21 b
22 dp
23 ho
24 ltd
25 net
26 pr
27 vg

Chromosome 3

41 cd
42 cu
43 e
44 gl^3
45 h
46 ru^g jv se by
47 se
48 st

Chromosome 4

60 ci ey^R
61 ey^D ci^D

Chromosome 1, 2

71 v; bw

Chromosomes 2, 3

72 bw; ss
73 bw; st
74 bw; ru h ri
75 c; e
76 cn; st
77 Pm dp b/cy sp^2;
Sb/D,Cx F

Chromosomes 2, 4

78 pr; Mal

Chromosomes 1, 2, 3

79 v; cn; st

Translocation

80 $\frac{\text{b pr tk}}{\text{T(Y;2)G}}$

LAFAYETTE, INDIANA: PURDUE UNIVERSITY
Department of Biological Sciences

Note: Stock list is the same as given in DIS-33 except:

Delete	Add
C-47 dp b Px^4/Gla	C-20 da/Ins SM1, al^2 Cy sp^2
H-59 y f:= and +; l(2)55i/Ins SM1, al^2 Cy sp^2; Sb/Ubx^{130} e^s; pol	

LAWRENCE, KANSAS: UNIVERSITY OF KANSAS
Department of Entomology

Note: Stock list unchanged. See DIS-30 p. 26.

LEXINGTON, KENTUCKY: UNIVERSITY OF KENTUCKY

Wild Stocks

Big Ridge, Tenn. (single female strain), 1948
Bikini Atoll (mass-inbred strain), 1947
Pine Ridge, Ky. (mass-inbred strain), 1954

MINNEAPOLIS, MINNESOTA: UNIVERSITY OF MINNESOTA
Departments of Zoology and Animal Husbandry

Note: Only unusual stocks are listed.

Equilibrium mutant segregating (EMS) populations: Non-inbred populations in which one or more mutants with visible effects are present and in which there is a near approach to linkage equilibrium with respect to each mutant locus relative to all other loci concerned. Each population was initiated by crossing mutant and wild type stocks and the derived populations have been reproduced by selecting 3:1 or 9:3:3:1 ratios so that the frequencies of the mutants are held at approximately .5. They were at generation 25 on November 25, 1960.

1. e x Wild Gilbert
2. bw x Wild synthetic
3. bw x Falcon Woods wild
4. al x Falcon Woods wild
5. e x bw
6. al x bw
7. al x e
8. th x e

OAK RIDGE, TENNESSEE: OAK RIDGE NATIONAL LABORATORY
Biology Division

Wild Stocks

a-1 Canton-S a-2 Oregon-R a-3 Oregon-R-C a-4 Swedish-c a-5 Samarkand

Normal X Chromosome

b-1 B/y f:=
b-2 car bb
b-3 Co/y w f
b-4 cx^{tg} t/FM1, y^{31d} sc^8 w^a lz^s B
b-5 dow/FM6, y^{31d} sc^8 dm B
b-6 ec dx
b-7 f
b-8 f BB/y f:=
b-9 f fu/ClB
b-10 fa
b-11 fa fa^{no} sn^3
b-12 fa N^{22a}/In(1)dl-49, y Hw m^2
b-13 fa N^{22c} sn^3/In(1)dl-49, y Hw m^2
b-14 fa rb
b-15 fa spl sn^3
b-16 fa^{no}
b-17 fa^{no} spl
b-18 fu^{59}/y f:=
b-19 l(1)J1 sc^{J1}/Del(1)24
b-20 kz/FM6, y^{31d} sc^8 dm B
b-21 m f car
b-22 N^{264-40}/In(1)dl-49, y Hw m^2 g^4
b-23 $N^{264-109}$/In(1)dl-49, y Hw m^2 g^4

b-24 N^{Co}/In(1)dl-49, y Hw m^2
b-25 nd
b-26 nd rb
b-27 ptg^3 v m g^2 sd f/y f:=
b-28 ras dy
b-29 rst^2/FM1, y^{31d} sc^8 w^a lz^s B
b-30 rux/FM6, y^{31d} sc^8 dm B
b-31 s
b-32 sc cv v eq
b-33 sc cv v f B/y f:=
b-34 sc ec cv cy^6 v g/In(1)dl-49, y Hw m^2 g^4
b-35 sc ec cv ptg^3 v/y v f car
b-36 sn^4
b-37 spl
b-38 spl cho^2
b-39 spl dm/y f:=
b-40 spl rb
b-41 sw
b-42 v
b-43 v f su^w-f
b-44 w
b-45 w^a
b-46 w^a fa
b-47 w^a fa rb
b-48 w^a fa spl
b-49 w^a fa^{no} rb
b-50 w^a fa^{no} spl
b-51 w^a fa^{no} spl rb/y w f
b-52 w^a nd rb
b-53 w^a spl
b-54 w^a spl rb
b-55 w^{ch} rb/y w f
b-56 w^e bb^1/y f:=
b-57 w^e dy/y w f
b-58 w^t fw
b-59 y
b-60 y inbred line A/sc^8·Y
b-61 y inbred line B/sc^8·Y
b-62 y ac sc pn/y f:=
b-63 y ac sc pn w rb cm ct^6 sn^3 ras^2 v dy g^2 f car/Ins(1)sc^{S1},dl-49,sc^{S1} v f car
b-64 y B/y f:=
b-65 y bb^{13a}/y w/sc^8·Y
b-66 y bb^{174}/y w/sc^8·Y
b-67 y bb^{1158}/y w/sc^8·Y
b-68 y bb^{1452}/y^2 su-w^a bb/sc^8·Y
b-69 y bb^{1456}/y^2 su-w^a w^a bb/sc^8·Y
b-70 y cv v f
b-71 y cy v f car
b72 y f^{36a}
b-73 y fa^n sn^3
b-74 y l^{259}/sc^8·Y/S-5
b-75 y l^{451}/FM6, y^{31d} sc^8 dm B
b-76 y Hw/Ins(1) sc^{S1L},S,sc^{8R},sc^{S1} w^a B sc^8
b-77 y N^{264-47}/In(1)dl-49,y Hw m^2 g^4
b-78 y $N^{264-103}$/In(1)dl-49, y Hw m^2 g^4
b-79 y $N^{264-107}$/In(1)dl-49, y Hw m^2 g^4
g-15 y sc w^{col} spl f/In(1)rst^3, rst^3 f
b-80 y v car/y f:=
b-81 y w bb
b-82 y w fa^{no}
b-83 y w fa^{no} sn^3
b-84 y w spl sn^3
b-85 y w^a
b-86 y w^a m f car
b-87 y w^a spl rb
b-88 y^2 cho^2
b-89 y^2 cv v f
b-90 y^2 spl
b-91 y^2 w^a w/y f:=
b-92 No. 1663 (Fahmy)
b-93 No. 1920 (Fahmy)

Chromosome II

c-1 ab^2/T(Y;2)E
c-2 ab^2 tom_4 bw sp^2/Ins(2L+2R)Cy, Cy dp^{Th} Bl L^4 sp^2
c-3 al b c sp^2
c-4 al dp b pr c px sp
c-5 al sp b pr Bl c px sp/SM1, al^2 Cy sp^2
c-6 b cn c bw
c-7 b pr c px sp
c-8 Bl L^2/SM5, al^2 Cy lt^v sp^2
c-9 bw
c10 bw^D
c-11 cn bw
c-12 fr^2 wt/Ins(2L+2R)Cy
c-13 ho
c-14 lt stw^3
c-15 M(2)1/In(2R)Cy
c-16 M(2)S5/Ins(2L+2R)Cy, Cy (L^4 sp^2?)
c-17 M(2)S10/Ins(2L+2R)Cy, Cy pr Dp(2:2)$4l^2$
c-18 ms cn bw/dp^{txI} Cy pr Bl lt cn^2 L^4 sp^2
c-19 net al ex ds S ast shv ho rub/SM1, al^2 Cy sp^2
c-20 Pin^{Yt}/Ins(2L+2R)Cy, Cy
c-21 pr cn ix/SM5, al^2 Cy lt^v sp^2
c-22 sp^2 bs^2
c-23 Sp J L^2 Pin/SM5, al^2 Cy lt^v sp^2
c-24 stw^3 c
c-25 vg

Chromosome III

d-1 Bd^G/In(3R)C, l(3)a
d-2 bx^{34e}
d-3 C(3)x/tra
d-4 ca
d-5 ca^{nd}/In(3LR)Ubx^{130}, M(3)1 Ubx^{130} e^s
d-6 cu kar
d-7 cy-c sbd^2
d-8 D^3 H/In(3L)P, Mé
d-9 Dl^3/In(3R)C, e
d-10 e
d-11 e^4 wo ro
d-12 e^s
d-13 e^s ca^{nd}/In(3R)C, Sb e l(3)e
d-14 Gl Sb/LVM
d-15 H^2/In(3R)Vno, Vno
d-16 jvl
d-17 Ki red/TM1, Mé ri
d-18 l(3)tra Sb/In(3LR)Ubx^{130}, Ubx^{130} e^s

d-19 M(3)S34/T(2;3)Mé
d-20 p^p bx sr e^s
d-21 pb/In(3LR)Cx
d-22 Pc/TM1, Mé ri
d-23 Pr/In(3R)C, e
d-24 R Ly/In(3L)P, gm
d-25 red
d-26 ro Bd ca/In(3R)C, l(3)a
d-27 ru
d-28 ru h th st cu sr e^s ca
d-29 ru h th st cu sr e^s Pr ca/TM1, Mé ri
d-30 ry^2
d-31 se
d-32 se ss k e^s ro
d-33 sr gl
d-34 ss^a
d-35 st
d-36 st C_3G ca/In(3LR)Ubx 130, Ubx^{130} e^s
d-37 st in ri p^p
d-38 p^p Ki
d-39 st sbd e^s ro ca
d-40 st sr e^s ro ca
d-41 ve h th

Chromosome IV

e-1 bt
e-2 bt^D/ci^D
e-3 Ce^2/spa^{Cat}
e-4 ci ey^R
e-5 ci gvl ey^R sv^n
e-6 ci^D/ey^D
e-7 sv^n
e-8 spa
e-9 pol

Multichromosomal

f-1 v/Y^{bb}; (1;Y)
f-2 br^3 dx^{st}; ed Su^2-dx (1;2)
f-3 In(1)w^{m4}; E-Var7/Ins(2L+2R)Cy, Cy (1;2)
f-4 lz^D/In(1)dl-49, w^a? m; Ins(2L+2R)Cy, Cy/In(2LR)Pm, al^4 ds^{33k} lt^- bw^{V1} (1;2)
f-5 v; In(2R)bw^{VDe1}/Ins(2LR)SM1, al^2 Cy sp^2 (1;2)
f-6 v f; In(2R)bw^{VDe1}/SM1, al^2 Cy sp^2 (1;2)
f-7 $Y^SX{\cdot}Y^L$, In(1)EN, Y^S B y f·Y^L/y v bb/O; bw (1;2)
f-8 $Y^SX{\cdot}Y^L$, In(1)EN, $Y^Sy{\cdot}Y^Ly^+$/y^2 su-w^a bb/Y; cn bw (1;2)
f-9 In(1)AM, y^2/FM6, y^{31d} dm B; SM1, al^2 Cy sp^2/Bl; In(3R)Vno, Vno/In(3LR)Ubx^{130} Ubx^{130} e^s (1;2;3)
f-10 Ins(1)dl-49, B^{M1}, sc v B^{M1}, SM5, al^2 Cy sp^2/Pm; R112 (1;2;3)
f-11 spl rb; Ins(2LR)Sm1, al^2 Cy sp^2/In(2LR)Pm, al^4 ds^{33k} lt^- bw^{V1}; C Sb/In(3LR) Ubx^{130}, Ubx^{130} e^s (1;2;3)
f-12 y; Ins(2L+2R)Cy, Cy/In(2LR)Pm, al^4 ds^{33k} lt^- bw^{V1}; Sb/In(3L)D, D (1;2;3)
f-13 y f:=/T(3;1)O-5/Y; SM1, al^2 Cy sp^2/In(2LR)Pm, al^4 ds^{33k} lt^- bw^{V1} (1;2;3)
f-14 $Y^SX{\cdot}Y^L$, $FR1^LC\text{-}2^R$, y; bw; st (1;2;3)
f-15 v; In(2R)bw^{VDe1}/SM1, al^2 Cy sp^2; M-4/ey^D (1;2;4)
f-16 y^2 v; bw; ci^D/ey^D (1;2;4)
f-17 y^2 v; bw; ey^D/simulans-4 (1;2;4)
f-18 y; bw; e; ci ey^R (1;2;3;4)
f-19 y f:=; bw; e; ci ey^R (1;2;3;4)
f-20 w; e (1;3)
f-21 y; ru h th st p^p cu sr e^s (1;3)
f-22 $Y^SX{\cdot}Y^L$,In(1)EN, Y^Sw y·Y^Ly^+/y^2 su-w^a w^a bb/O; Sb/In(3LR)Ubx^{130}, Ubx^{130} e (1;3)
f-23 XY, w/y^2 w^a bb/O;Ubx 130e/Sb (1;3)
f-24 sc cv v f B; ci ey^R (1;4)
f-25 w cv v f; sv^n/sv^n/sv^n (1;4)
f-26 y; sv^n (1;4)
f-27 y f:=; ci ey^R (1;4)
f-28 $Y^SX{\cdot}Y^L$, In(1)EN, y B/y^2 su-w^a w^a bb/O; sv^n (1;4)
f-29 al; ru (1;3)
f-30 b pr Bl/SM1, al^2 Cy sp^2; In(3R)Vno, Vno/In(3LR)Ubx^{130}, Ubx^{130} e (2;3)
f-31 bw; st(2;3)
f-32 Ins(2L+2R)Cy, Cy/In(2LR)Pm, al^4 ds^{33k} lt^- bw^{V1}; Sb/In(3LR)DcxF, D (2;3)
f-33 SM1, al^2 Cy sp^2/In(2LR)Pm, dp b ds^{33k}; Sb/Ins(3LR)Ubx^{130}, Ubx^{130} e^s (2;3)
f-34 stw^3 c; st (2;3)
f-35 bw; ci sv^n (2;4)
f-36 bw; ci^D/ey^D (2;4)
f-37 bw; ci^D/simulans-4 (2;4)

Inverted X Chromosomes

g-1 In(1)AM/T(1;3)65 y
g-2 In(1)AB, sc/y f:=
g-3 In(1)AB, sc cv
g-4 In(1)AB, y f
g-5 In(1)B^{M1}, cm/y f:=
b-9 In(1)CI, sc l t^2 v sl B (ClB)
g-6 In(1)dl-49, y fa^n
b-12 In(1)dl-49, y Hw m^2
b-22 In(1)dl-49, y Hw m^2 g^4
i-8 In(1)dl-49, y Hw m^2 g^4 f^5
g-7 In(1)dl-49, y v f car/y f:=
g-8 In(1)dl-49, v^{Of} f
g-9 In(1)dl-49, ty-1 bb^1/y v f car
g-10 In(1)dl-49, y w B

g-11 In(1)dl-49, y w lz^s/y f:=
g-29 In(1)dl-49, y w lz^s bb/In(1)sc^{L8}, sc^{L8} car m w^a
g-12 In(1)EN, y/y f:=
g-13 In(1)EN, y bb/sc^8.Y
g-14 In(1)rst^3, rst^3
g-15 In(1)rst^3, rst^3 f/y sc w^{col} spl f
g-16 In(1)rst^3, w^{48-3} rst^3 v f/y f:=
g-17 In(1)rst^3, y rst^3 car bb?/RA, y^2 w^a M-5/Y
g-18 In(1)sc^4, y sc^4
g-19 In(1)sc^4, y sc^4 cv v f
g-20 In(1)sc^{4L}, sc^{8R}, y sc^{4+8} cv v f/y f:=
g-21 In(1)sc^8, sc^8
g-22 In(1)sc^8 v f car
g-23 In(1)sc^8, sc^8 cv v f/In(1)dl-49, y Hw m^2 g^4
g-24 In(1)sc^8, y^{31d} sc^8 w^a
g-25 In(1)sc^8, y^{31d} sc^8 cv v f/y f:=
g-26 In(1)sc^8 (c·o·J-3^L), y^- ac^- sc^8 w^a f/RA, y ac sc Pn---In(1)sc^8·/y sc^8.Y
g-27 Ins(1)sc^8EN, y^+ f y/y f:=
g-28 Ins(1)sc^8EN, y^+ f y/sc^8.Y/y w
g-29 In(1)sc^{L8}, sc^{L8} w^a m car/In(1)dl-49, y w lz^s bb
g-30 In(1)sc^{L8l}, sc^{S1R}, sc^{L8} w^a m car/In(1)dl-49, y w lz^s bb
g-31 In(1)sc^{S1R}, sc^{8R}, sc^{S1} w^a m car sc^8
g-32 In(1)sc^{S1L}, sc^{4R}, sc^{S1} cv v B/y w/sc^8.Y
g-33 In(1)sc^{S1L}, sc^{4R}, sc^{S1} w^a m car y f:=
n-4 In(1)w^{m4}, w^{m4}
p-17 In(1)w^{m4L}, rst^{3R}, y w^- rst^3
g-34 In(1)y^{3P}, y^{3P} B
g-35 In(1)y^4, y^4
g-36 In(1)sc^{V1} y^+/Ins(1)sc^8, dl-49, y^{31d} y f B
g-37 In(1)sc^{V1}, y v·sc^{V1} y^+/y^2 w^a bb/sc^8.Y
g-38 Ins(1)dl-49, B^{M1}, sc v
g-39 Ins(1)sc^{4L}, AB, sc^{8R}, y sc^{4+8}/y f:=
b-74 Ins(1)sc^{4L}, S, sc^{8R}, y sc^{4+8} w^a B(S-5) /y l^{259}/sc^8.Y
i-9 Ins(1)sc^7, AM, sc^7
b-29 Ins(1)sc^8, dl-49, y^{31d} sc^8 w^a lz^s B(FM1)
g-40 Ins(1)sc^8, dl-49, sc^8 v f/y f:=
g-36 Ins(1)sc^8, dl-49, y^{31d} sc^8 v f B
h-37 Ins(1)sc^8, dl-49, 3C-4EF, y^{31d} sc^8 dm B (FM4)
g-57 Ins(1)sc^8, dl-49, 3C-4EF, 15DE-20, y^{31d} sc^8 dm B (FM6)
g-41 Ins(1)sc^8 (c·o·X J-3), S, y^- ac^- w^a sc^8/sc^8.Y
g-42 Ins(1)sc^{8L}, dl-49, sc^{S1R}, y sc- v B f/y l^{259} w m f/sc^8.Y
g-43 Ins(1)sc^{8L}, S, y^{3P}, y^{S1} sc^8 y^{3P}
g-44 Ins(1)sc^{L8L}, sc^{8R}, sc^{L8+8} cv v car/y w/sc^8.Y
g-49 Ins(1)sc^{S1}, dl-49, sc^{S1} v/y f:=
b-63 Ins(1)sc^{S1}, sl-49, sc^{S1} v f car
g-46 Ins(1)sc^{S1L}, AB, sc^{4R}, sc^{S1} w^a car/y f:=
g-47 Ins(1)sc^{S1L}, dl-49, sc^{8R}, sc^{S1+8} w m/w sn bb
g-48 Ins(1)sc^{S1L}, dl-49, sc^{8R}, sc^{S1+8} v B car/ y f:=
g-49 Ins(1)sc^{S1L}, dl-49, sc^{8R}, y sc^{S1+8} w^a v B/y f:=
g-50 Ins(1)sc^{S1L}, dl-49, sc^{8R}, y sc^{S1+8} w^a v f/y f:=
g-57 Ins(1)sc^{S1L}, S, sc^{8R}, sc^{S1+8} w^a B (M-5)
g-52 Ins(1)sc^{S1L} , S, sc^{8R}, sc^{S1+8} w^a B bb^1/y f:=
g-53 Ins(1)sc^{S1L}, S, sc^{8R}, y^2 sc^{S1+8} w^a B/sc^8.Y
g-54 Ins(1)sc^{S1L}, S, sc^{8R}, y^2 sc^{S1+8} w^a B
g-55 Ins(1)sc^{S1L}, S, sc^{8R}, y^{54k7} ac^{54k7} sc^{S1+8} w^aB/y f:=
g-56 Ins(1)y^{3PL}, S, sc^{S1R}, y^- ac^- sc^-/ y f:=; Cy/sc^{19i}
g-57 In(1)481(12E-F;14B), y bb^{1481}/FM6/sc^8.Y
g-58 In(1H)59(3-4), y l^{59}/y w/sc^8.Y
g-59 In(1H)132(4E) y l^{132}/y w/sc^8.Y
g-60 In(1H)146(4D), y l^{146}/y w/sc^8.Y
g-61 In(1H)227(1F), y l^{227}/y w/sc^8.Y
g-62 In(1H)231(5C-D), y l^{231}/y w/sc^8.Y
g-63 Inp(1)139(3C), y w^{m139} l^{139}/FM6, y^{31d} sc^8 dm B
g-64 Inp(1)139, w^{m139}, rst^m l^{139}/y w f/Y/Y
g-65 In(2LR)lt^{m3}(60D)/SM5
g-66 In(2LR)lt^{m12}(60D)/SM5
g-67 In(3R)18/Xa
g-68 In(3R)112

Deficiencies and Duplications

p-1 Df(1)N^8/In(1)dl-49, y Hw m^2
b-29 Df(1)rst^2/FM1, y^{31d} sc^8 w^a lz^s B
g-42 Df(1)sc (see Ins(1)sc^{8L}, dl-49, sc^{S1R})
p-2 Df(1)sc^{10-1}/y Hw
p-17 Df(1)w (see In(1)w^{m4L}, rst^{3R})
p-3 Df(1)w^{258-45}/FM4, y^{31d} sc^8 dm B
p-3a Df(1)w^{258-45}, y; Dp(1;3), w^{Vco}/y w f
p-3b Df(1)w^{258-45}, y spl dm; Dp(1;3), w^{Vco}/y w f
p-4 Df(1)w^{258-48}, y sc^5 spl; Dp(1;3), w^{Vco}/y f:=
p-5 Dp(1;f)3 = Del(1)3/y/XY^L.Y^S, y l^{259} w Y^L.Y^S
p-6 Dp(1;f)18 - Del(1)18/y v f/XY^L.Y^S, y l^{259} w Y^L.Y^S
b-19 Dp(1;f)24 = Del(1)24
p-7 Dp(1;f)52 = Del(1)52/y v f/y l^{259} w Y^L.Y^S
p-8 Dp(1;f)112 - Del(1)112/y v f/y l^{259} w Y^L.Y^S
p-9 Dp(1;f)122 - Del(1)122/y v f/y l^{259} w Y^L.Y^S
p-10 Dp(1;f)164 - Del(1)164/y v f/y l^{259} w Y^L.Y^S

p-11 Dp(1;f)1492 = Del(1)1492/sc^{53k}
p-12 Dp(1;f)1514 = Del(1)1514/sc^{53k}
p-13 Dp(X^c;f)6 = Del(X^c)6/RA l(1)J1·sc^8/ $Y^S X·Y^L$, In(1)EN, y
p-14 Dp(1;1)B^S(RAG), B^S--In(1)sc^8·/Ins(1) sc^7, AM
p-15 Dp(1;1)B^S(TMG), In(1)sc^4·B^S, y sc^4 m f·B^S/Ins(1)sc^7, AM
p-16 Dp(1;1)B^S(TMG), In(1)sc^{8L}, X^{c2R}·B^S, f·B^S/X^D/$B^S Y^L$·Y^S
h-3 Dp(1;2)sc^{19i}
p-17 Dp(1;2R)w^{51b7}/y w f/In(1)w^{m4L}, rst^{3R}, y w^- rst^3
p-18 Dp(1;3)w^{49a7} (Spotter)
p-19 Dp(1;3)51 - T(1;3)51/y v f/XY^L·Y^S, y l^{259} w Y^L·Y^S
p-20 Dp(1;3)142 = T(1;3)142/y/XY^L·Y^S, y l^{259} w Y^L·Y^S
p-21 Dp(1;3)sc^{J4}/y v f/0 ?/XY^L·Y^S, y l^{259} w Y^L·Y^S
p-22 Dp(1;4)w^{51c20}/y w f:=/In(1)w^{m4L}, rst^{3R} car rst^3
p-23 Dp(1;4)174 = T(1;4)174/y v f/y l^{259} w Y^L·Y^S
p-24 Df(4)M-4/ey^D

Translocated Chromosomes

h-1 T(1;2)459, y l^{459}/FM6, y^{31d} sc^8 dm B
h-2 T(1;2)Bld/ClB
h-3 T(1;2)sc^{19}/y f:=; fes sc^{19i} b pr/ Cy dp^{TH} pr
h-4 T(1;1H)25(20), y l^{25}.FM6
h-5 T(1;1LH)150(16-17), y l^{150}/FM6
h-6 T(1;2LH)219(10A), y l^{219}/FM6
h-7 T(1;2RH)75(20), y l^{75}/FM6
h-8 T(1;2RH)135(18-19), y l^{135}/FM6
h-9 T(1;2;3)220(14A;50A;75), y l^{220}/ FM6
h-10 T(1;2;3;4)454, y l^{454}/FM6
h-11 T(1;2H)361(20), y l^{361}/FM6
h-12 T(1;3H)453(12D), y l^{453}/FM6
h-13 T(1;3H)463(20), y l^{463}/FM6
h-14 T(1;2LH)163(17A-A), y l^{163}/FM6
h-15 T(1;3LH)455(3C), y l^{455}/FM6
h-16 T(s;3RH)3(3-4), y l^3/FM6
h-17 T(1;3RH)129(18B), y l^{129}/FM6
h-18 T(1;4)A7, y w/y^2 su-w^a w^a bb
h-19 T(1;4)A13(18C5)
h-20 T(1;4)A17(8A2)/y f:=
h-21 T(s;4)A17(8A2), y cv/y f:=
h-22 T(1;4)A19
h-23 T(1;4)A20/y f:=
h-24 T(1;4)B^S(16A1), B^S/y f:=
h-25 T(1;4)B^S(16A1), B^S car/y f:=
h-26 T(1;4)B^S(16A1), y B^S/y f:=
h-27 T(1;4)B^S(16A1), y cv v B^S/y f:=
h-28 T(1;4)B^S(16A1), y^2 cv v B^S car/ y f:=
h-29 T(1;4)B^{SL}; 11^R, y/y w
h-30 T(1;4)e15
h-31 T(1;4)h4
h-32 T(1;4)h6
h-33 T(1;4)11(15A), y l^{11}/y w/sc^8·Y
h-34 T(1;4)In(1)sc^{4L},sc^{8R}, y sc^{4+8} w^a m car/y f:=
h-35 T(1;4)w^{258-18}
h-36 T(1;4)w^{m5}(3C3), w^{m5}
h-37 Ts(1;4)w^{m5},B^S(3C3,16A1), w^{m5} v B^S/ FM4, y^{31d} w dm f
h-38 T(1;4)(4C3)/y f:=
h-39 T(1;4)(13B809)/y f:=
h-40 T(XY^L·Y^S;4)B^S(16A1), X^D,$B^S Y^L$·Y^S/ y v bb:=/0
h-41 T(Y;2)E(36D)/y^2 su-w^a w^a bb
h-42 T(Y^S;4)
h-43 T(2;1)223(41-50;14),y l^{223}/FM6
h-44 T(2;3)bw^{V4}; bw^{V4}/Cy
h-45 T(2;3)bw^{V5}; bw^{V5}/Cy
h-46 T(2;3)lt^{m7}(98C), lt^{m7}/SM5
h-47 T(2;3)S^M; S^M Cy/vg^{nw}
h-48 T(2;3)S^M, S^M Cy C_3G Sb Ubx/st C_3G ca
h-49 T(2;3)Xa/In(3LR)Ubx^{130}, Ubx^{130} e^s
h-50 T(3;1)05, D
h-51 T(3;4)86D, bx^{34e} e^4
h-52 T(3;4)88B, Ubx/ey^D
h-53 T(3;4)89E, ss bx bxd/ey^D

Closed X Chromosomes

i-1 X^c, y/y f:=
i-2 X^{c2} (ET), +/M-5
i-3 X^{c3} (KOA), +/M-5
i-4 X^{c2}, w^{col} 491/In(1)dl-49, y Hw m^2 g^4
i-5 X^{c2},w^{spont} v f/RM, Ins(1) sc^{S1L}, S, sc^{8R}, sc^{S1} w^a sc^8/Y
i-6 X^{c2}, y^{49}
i-7 X^{c2}, y f car
i-8 X^{c2}, In(1)w^{vc} (stable), w^{vc}/In(1) dl-49, y Hw m^2 g^4 f^5
i-9 X^{c2}, In(1)w^{vc} (stable), w^{vc}/Ins(1) sc^7, AM
i-10 X^{c2}, In(1)w^{vc} (stable), w^{vc} f/y f:=
i-11 X^{c2}, In(1)AB, +/y f:=
i-12 X^{c56K-1} (from RR L-26), y cv v f/ y f:=/sc^8·Y
i-13 X^{c56k-4} (from RRL-26), y ? ?/In(1) dl-49, y w lz^s/Y ? ?

X Chromosomes with a Y Arm Attached

j-1 X·Y^L (A-2), y w·Y^L?y/Y"
j-2 X·Y^L (C-2), y cv v f car bb^-·Y^L/RA, (ND-27) v f/Y"
j-3 X·Y^L (C-2), y w bb^-·Y^L/y/Y"
j-4 X·Y^L (U-8e), sc cv v f·Y^L/y/Y"
j-5 X·Y^L (U-8e), y w·Y^L/y/Y"
j-6 X·Y^L (Stern), g^2 B·Y^L/y/Y"
j-7 X·Y^L, y cv v f car ·Y^L/y/Y"
j-8 X·Y^L, y v f bb(bb^+·)Y^L/y f:=/sc^{V1}·Y^S
j-9 X·Y^L, In(1)sc^{8L},EN^R, y^+ car f v cv y·Y^L/y/Y"
j-10 X·Y^L, In(1)sc^{8L}, EN^R, y^{31d} f v cv·Y^L/ Y"

j-11 X·Y^L (K-7), In(1)sc^{8L},EN^R, y^+ f y·Y^L/y/Y"
j-12 X·Y^L (K-7), In(1)sc^{8L},EN^R, y^+ f v cv y·Y^L/y/Y"
j-13 X·Y^L (P-8b), In(1)sc^{8L},EN^R, y^+ f y·Y^L/y/Y"
j-14 X·Y^S (A-3), y w·Y^S/y v f/Y^{Lc}
j-15 X·Y^S (A-3), sc cv v·Y^S/y v f/Y^{Lc}
j-16 X·Y^S (Muller), y w·Y^S/y v f/Y^{Lc}
j-17 X·Y^S (U-8c), y w·Y^S/y v f/Y^{Lc}
j-18 X·Y^S (U-8c), y cv v·Y^S/Y^{Lc}
j-19 X·Y^S (PDP), y^2 cv v·Y^S/In(1)dl-49, y Hw m^2 g^4/Y^{Lc}
j-20 X·Y^S (P-8b), In(1)sc^{8L},EN^R, y^+ f y·Y^S/y v f/Y^{Lc}
j-21 Y^SS·, (FR-1), Y^S y cv v f/y f:=/Y
j-22 Y^SX·, (Fr-1^L,In(1)p^R), Y^S y cv v f·y^+/Y^{Lc}
j-23 Y^SX·, (Fr-1), Y^S y m f car/y v f:=/Y^{Lc}
j-24 Y^SX· (P-0),In(1)EN, Y^S y/y w/Y
j-25 Y^SX· (P-7), In(1)EN, Y^S y f/y v f/Y

Attached XY Chromosomes

m-1 XY^L·Y^S (2-10T13 Parker), y^2 su-w^a w^a Y^L·Y^S/y/Y
m-2 XY^L·Y^S (2-10T15 Parker), y^2 su-w^a w^a Y^L·Y^S/y/Y
m-3 XY^L·Y^S 108-9 Parker), y^2 su-w^a w^a Y^L·Y^S/y v bb/0
m-4 XY^L·Y^S (112-17 Parker), y^2 su-w^a w^a Y^L·Y^S/y v bb/0
m-5 XY^L·Y^S (127-29 Parker), y^2 su-w^a w^a Y^L·Y^S/y v bb/0
m-6 XY^L·Y^S (129-11 Parker), y^2 su-w^a w^a Y^L·Y^S/y v bb/0
m-7 XY^L·Y^S, y l(1)259 w Y^L·Y^S/y/Dp(1;f)167
m-8 XY^S·Y^L (110-8 Parker), y^2 su-w^a w^a Y^S·$Y^L$$y^+$/y v bb/0
m-9 XY^S·Y^L (115-9 Parker), y^2 su-w^a w^a Y^S·Y^L y^+/y v bb/0
m-10 XY^S·Y^L (129-16 Parker), y^2 su-w^a w^a Y^S·Y^L y^+/y v bb/0
m-11 Y^SX·Y^L (FR 1^L,C-2R), Y^S y bb$^-$·Y^L/y^2 su-w^a w^a bb/0
m-12 Y^SX·Y^L (FR-1^L,U-8d^R), Y^S y w^a cv v f·Y^L/y^2 su-w^a w^a bb/0
m-13 Y^SX·Y^L, In(1)EN, Y^S B f v y·Y^L y^+/y v bb/0
m-14 Y^SX·Y^L, In(1)EN, Y^S B f v w y·Y^L y^+/y^2 su-w^a w^a bb/0
m-15 Y^SX·Y^L, In(1)EN, Y^S B y·Y^L/y^2 su-w^a w^a bb/0
m-16 Y^SX·Y^L, In(1)EN, Y^S y·Y^L/y^2 su-w^a w^a bb/0
m-17 Y^SX·Y^L, In(1)EN, Y^S v cv y·Y^L y^+/y^2 su-w^a w^a bb/0
m-18 Y^SX·Y^L, In(1)EN, y^+ Y^S y·Y^L y^+/y^2 su-w^a w^a bb/0
m-19 Y^SX·Y^L, In(1)EN, Y^S y·Y^L y^+
m-20 Y^SX·Y^L, Ins(1)EN,17, Y^S B f v y·Y^L y^+/y f:=/Y
m-21 Y^SX·Y^L, Ins(1)EN,18, Y^S B f v y·Y^L y^+/y f:=/Y
m-22 Y^SX·Y^L, Ins(1)EN,20, Y^S B f v y·Y^L y^+/y f:=/Y
m-23 Y^SX·Y^L, Ins(1)EN,24, Y^S B f v y·Y^L y^+/y f:=/Y
m-24 Y^SX·Y^L, Ins(1)EN,24^L,A-2^R, Y^S y v·Y^L/y^2 su-w^a w^a bb/0
m-25 Y^SX·Y^L, Ins(1)EN,32, Y^S B f v y·Y^L y^+/y f:=/Y
m-26 Y^SX·Y^L, Ins(1)EN,39, Y^S B f v y·Y^L/y f:=/Y
m-27 Y^SX·Y^L, Ins(1)EN,42, Y^S B f v y·Y^L y^+/y f:=/Y
m-28 Y^SX·Y^L, Ins(1)EN,44, Y^S B f v y·Y^L y^+/y f:=/Y
m-29 Y^SX·Y^L, Ins(1)EN,46 Y^S B f v y·Y^L y^+/y^2 su-w^a w^a bb/0
m-30 Y^SX·Y^L, Ins(1)EN,dl-49, Y^S car f v y·Y^L/y^2 su-w^a w^a bb/0
m-31 Y^SX(Y^L·) (FR-1^L,3-18^R), Y^S y (Y^L·bb^+) RA,y^2 su-w^a w^a---M-5/Y
m-32 Y^SX(Y^L·) (FR-1^L,118-2b^R#1), Y^S y (Y^L·bb^+)/y^2 su-w^a w^a bb/0
m-33 Y^SX(Y^L·) (FR-1^L,118-2b^R#20), Y^S y cv (Y^L·bb^+)/y^2 su-w^a w^a bb/0
m-34 Y^SX(Y^L·) (FR-1^L,118-2b^R#?), Y^S y cv (Y^L·bb^+)/y^2 su-w^a w^a bb/0
m-35 Y^SXY^L· (FR-1^L,174-13^R), Y^S y cv v bb Y^L·/RA y^2 su-w^a w^a---M-5/Y
m-36 Y^SXY^S·Y^L (FR-1^L,115-9^R), Y^S y cv Y^S·Y^L y^+/y^2 su-w^a w^a bb/0

Compound X Chromosomes

k-1 RA, l(1)J1 sc^{J1}--In(1)sc^8·/XY^L·Y^S, y l^{259}w Y^L·Y^S/y sc^8·Y
j-2 RA(ND-27), sc v f--In(1)sc^8, f v sc^8·/X·Y^L(C-2), y cv v f car bb$^-$·Y^L/Y"
n-12 RA, y--In(1)sc^8·/Y^SX·Y^L, In(1)EN,Y^SB y·Y^L/y^+ ac^+·Y^L
g-26 RA, y ac sc pn--In(1)sc^8·

k-2 RA (ND 9-3), sc--In(1)sc^8·/$Y^SX·Y^L$, In(1)EN,Y^SB f y·Y^L/y^+ ac^+·Y^L
k-3 RA, y--In(1)sc^{8L}, EN^R·Y^L/$Y^SX·Y^L$, In(1)EN, Y^SB y·Y^L/y^+ ac^+·Y^L
k-4 RA, In(1)AB, y--In(1)sc^8·/$Y^SX·Y^L$, In(1)EN·Y^SB y·Y^L/y^+ ac^+·Y^L
n-14 RA (Muller), In(1)dl-49, y w f--In(1)sc^8? f sc^8·/XY^S, y w·Y^S/Y^L·bb^+ ac^+ y^+
p-22 RA(Muller), In(1)dl-49, y w f--In(1)sc^8?, f w sc^8·/Dp(1;2R)w^{51b7}/Y/In(1)w^{m4L}, rst^{3R}
k-5 RA (ND-33), y f car--In(1)sc^8, car f sc^8·/$Y^SX·Y^L$, In(1)EN,Y^SB y·Y^L/y^+ ac^+·Y^L
m-31 RA, y^2 su-w^a w^a--Ins(1)sc^{S1L}, S, sc^{8R}, B w^a sc^8·/$Y^SX(Y^L·)$, Y^Sy(Y^L·bb^+)/Y
n-6 RA,y l^{259} w--In(1)sc^8·/y l^{259} w Y^L·Y^S/ y sc^8·Y
h-7 RA·Y^L, +--In(1)sc^{8L}, EN^R, y·Y^L y^+/$Y^SX·Y^L$, In(1)WN, Y^SB y·Y^L/y^+ ac^+·Y^L
j-1 RM, y/X·Y^L(A -2), y w·Y^L/Y"
m-9 RM, y v bb
n-13 RM, y v f/X·Y^S,y w·Y^S/ac^+ y^+·Y^L
b-35 RM, y v f car
n-16 RM, y w/X·Y^S, y w·Y^S/Y^L bb^+ ac^+ y^+
n-17 RM, y^2 su-w^a w^a bb/X·Y^S, y w·Y^S/Y^L bb^+ ac^+ y^+
i-5 RM, Ins(1)sc^{S1L}, S, sc^{8R}, sc^S w^a sc^8/Xc^2, w^{spont} v f
k-8 RM (13-0-15=XY^L·X), y^2 su-w^a w^a bb Y^L/y^2 su-w^a w^a bb? bb^+/$Y^SX·Y^L$, In(1)EN, Y^SB y·Y^L
k-9 RM (15-DRP=XY^L·Y^LX), y^2 su-w^a w^a bb Y^L/y^2 su-w^a w^a bb? $Y^?$ bb^+/$Y^SX·Y^L$, In(1)EN, Y^SB f v y·$Y^L$$y^+$
k-10 RM, In(1)EN, Y^S y/O.Y^SX·Y^L, Ins(1)EN, dl-49, Y^S car f v y·Y^L
k-11 RM(L-6), Y^SX·Y^L, In(1)EN, Y^SB y·Y^L, originally In(1)sc^{S1L}, EN^R, sc^{S1}car m y/ In(1)sc^8, f v cv sc^8
k-12 RM (L-26), Y^SX·Y^L, In(1)EN, Y^SB y·Y^L, originally In(1)sc^{S1L}, EN^R, sc^S car m y? In(1)sc^8, f v cv sc^8
k-13 RM (TAX), In(1)y^4, w^a y^4/y v f/$Y^SX·Y^L$, In(1)EN, Y^SB y·Y^L/O
k-14 TM Hw f), originally y Hw v f·y^+ cv f y^+/X·Y, y B
k-15 TM, In(1)dl-49·In(1)EN^R, sc^{4L}, y v f?·y m sc^4 y (stabilized by a 1-2 crossover)y B/Y

Altered Y Chromosomes

n-1 sc^8·Y (y^+ ac^+ Y^L·bb^+ Y^S)/y w/y
n-2 sc^8·Y:bw^+ (Y^L bw^+·bb^+ Y^S ac^+ y^+)/y f:=/y v f
n-3 sc^8·Y (y ac^+ Y^L·bb^+ Y^S)/Muller-5
n-4 Ybb-/In(1)w^{m4}, w^{m4}
n-5 Ybb-/y^2 eg
n-6 YB^S (B^S Y^L·bb^+ Y^S)/y^2 su-w^a w^a bb
n-7 YB^Ssc^8 (B^S Y^L·bb^+ Y^S y^+)/y v; bw
n-8 YSu-Var/In(1)w^{m4}, w^{m4}
n-9 Y:bw^+ (Y^L bw^+·bb^+ Y^S)/y v; bw
n-10 Y y^{31d} B^S/y w f/Y^S y cv v f·
n-11 Y^c:bw^+ (MYR)/y v; bw
n-12 y^+ ac^+ Y^L· (FR-2)/$Y^SX·Y^L$, In(1)EN, Y^SB y·Y^L/RA, y--In(1)sc^8
n-13 Y^L·ac^+ y^+ (sc^8EN c·o·Y B-2)/X·Y^S, y w·Y^S/y v f
n-14 Y^L·bb^+ ac^+ y^+ (sc^8EN c·o·Y T-0)X·Y^S, y w·Y^S/y f:=
n-15 Y^L·bb^+ ac^+ y^+ (sc^8 c·o·Y T_o)/X·Y^S, y w·Y^S/y f:=
n-16 Y^L·bb^+ ac^+ y^+ (sc^8EN c·o·Y U-8)/X·Y^S,y w·Y^S/y w
n-17 Y^L·bb^+ sc^{S1} ac^+ y^+ (sc^S c·o·Y CY9)/X·Y^S, y w·Y^S/y^2 su-w^a w^a bb
n-18 Y^L·bb^+ sc^{S1} ac^+ y^+ (sc^S c·o·Y EY80)/X·Y^S, y w·Y^S/y v f
j-15 Y^{Lc}/X·Y^S(A-3) sc cv v·Y^S/y v f
j-1 Y^S·Y^S (Y" Stern)/y/X·Y^L(A-2),y w·Y^L
j-8 Y^S·sc^{V1} ac^+ y^+ (sc^{V1}·Y^S)/X·Y^L, y v f bb(bb^+·)Y^L/y f:=

PHILADELPHIA, PENNSYLVANIA: THE INSTITUTE FOR CANCER RESEARCH
Department of Chemotherapy

Wild Stocks

a1 + Amherst 3 (homoz. Singh, 1939)
a2 + Canton-S, A (iso, 1952)
a3 + Crimea
a4 + Florida-9
a5 + Lausanne-S
a6 + Oregon R
a7 + Samarkand
a8 + Seto, Japan
a9 + Swedish-b-6
a10 + Urbana-S
a11 + Wageningen

Chromosome 1 (X)

b1 ac^3 w^a.Dp(sc^{V1} y^+) & y f:=
b2 amx/FM3, y^{31d} sc^8 dm B l
b3 amx^{55}
b4 Ax
b5 B
b6 B Bx^r car & y f:=
b7 B car su^w-f & y w f:=
b8 B^{M2} f^{B27}/ClB (mosaic in f/f^{27})
b9 Bg B/InAM
b10 bi ci^6 g^2
b11 bo
b12 br
b13 br w^e ec rb t^4/FM1, y^{31d} sc^8 w^a lz^s B
b14 Bx
b15 Bx^2
b16 Bx^3
b17 Bx^J
b18 Bx^{r49k} & y f:=
b19 car
b20 car bb
b21 cm
b22 cm ct^6
b23 cm ct^6 sn^3 & y w f:=
b24 cs^{53} & y w bb·=
b25 ct^6 v dy g f/InA99
b26 ct^n oc/FM1, y^{31d} sc^8 w^a lz^s B
b27 cv f
b28 cx
b29 cx^{tg} t/FM1, y^{31d} sc^8 w^a lz^s B
b30 Df(1)259-4/FM4, y^{31d} sc^8 dm B
b31 Df(1)260-1/FM4, y^{31d} sc^8 sm B
b32 Df(1)g^1 f B/InAM
b33 Df(1)svr, Dp(1;f)101 (Dp. het. or hom.)
b34 dor/ClB
b35 dow/y Hw In49 m^2 g^4
b36 dm & y f:=

b37 Dp(1;f)101 In(1)sc^8, Df(0+ac)w^a sc^8; Dp(1:f)101
b38 Dp(1;f)107 In(1)sc^8, Df(0+ac)w^a sc^8; Dp(1;f)101
b39 Dp(1;f)118 In(1)sc^8, Df(0+ac)w^a sc^8; Dp(1;f)118
b40 Dp(1;f)135 In(1)sc^8, Df(0+ac)w^a sc^8; Sp(1;f)135
b41 Dp(1;f)135, y^2; y w bb
b42 Dp(1;f)X^{c2} Dp(s;f)X^{c2}/y l(1)7/y l(1)7
b43 Dp(1;f)z^9 Dp(1;f)z^9; Df(1)sc^{J4R} & y f:=
b44 Dp(1;1)112 y f, Dp(1;1)112 (homozygous stock)
b45 Dp(1;1)Co
b46 Dp(1;1)Co, Df(1)rst^2 & y w bb·=
b47 Dp(1;Y^L)sc^{S1} sc^{S1}.Y^L/y·Y^S;y f:=; cn bw; (e/+)
b48 Sp(1;3)126 v f; Dp(1;3)126/Payne, Dfd ca
b49 Dp(1;3)sc^{J4} Dp(1;2)sc^{J4}/Df(1)sc^8, w^a
b50 dy

b51 ec
b52 ec ct^6 v g^3/ClB
b53 ec dx
b54 ec dx/y su-Hw Hw^2 In49 m^2 g^4
b55 Ext/FM6, y^{31d} sc^8 dm B
b56 f B car su^w-f & y f:=
b57 f B odsy car
b58 f B odsy f^{+ih} & y f:=
b59 f B^3 & y f:=
b60 f B^i (Luce 436.1) & y f:=
b61 f B^iB^i & y f:=
b62 f BB & y f:=
b63 f BB^{36b} & y f:=
b64 f fu/ClB
b65 f fu & y f:=
b66 f od car
b67 f^5 odsy f^{+ih} & y w f:=
b68 f^5 su^w-f
b69 f^{36a}
b70 f^{36a} odsy f^{+ih} & y f:=
b71 f^x car & y f:=
b72 fa
b73 flp
b74 (Triploid) FM4, y^{31d} sc^8 dm B/y^2 sc w^a ec·=
b75 fo
b76 g^2
b77 g^2 pl.FM3, y^{31d} sc^8 dm B l
b78 g^2 ty & y·=
b79 g^{im}/y sc^{S1} B InS
b80 g^x,Inh & y f:=
b81 gg^2/FM6, y^{31d} sc^8 dm B
b82 gg^3
b83 gt bb^{11}/ClB
b84 gt v
b85 gt w^a
b86 gt w^a (Oregon-R)
b87 Hw^{49c}/FM1, y^{31d} sc^8 w^a lz^s B
b88 if^3
b89 InAB & y f:=
b90 In49 B^{M1}
b91 In49 v Fl g & y w f:=
b92 In49 lz^s & y f:=
b93 In49 m v sn^{x2} g/y ClB
b94 In49 oc ptg & y f:=
b95 In49 sn^{x2} & y f:=
b96 In49 v sn^{x2} B &y f:=
b97 In49 y^{0f}
b98 In(X^{c2})w^{vc}/y Hw In49 m^2 g^4 f^5 (ring stabilized)
b99 kz
b100 l(1)7/FM6, y^{34} sc^8 dm B
b101 lh B car bb & y f:=
b102 lz/FM3, y^{31d} sc^8 dm B l
b103 lz^3 & y f:=
b104 lz^3 m & y w f:=
b105 lz^{34k} & y f:=
b106 lz^{37h}
b107 lz^{48f} & y f:=
b108 lz^{BS} lz^{46f} ras^4 v & y f:=
b109 m
b110 m^D/FM3, y^{31d} sc^8 dm B l
b111 ma-l & y f:=
b112 M(1)o f/InAM
b113 M(1)Sp/InAM
b114 na & y f:=
b115 ny f/FM1, y^{31d} sc^8 w^a lz^s B;(ri)
b116 N^8/y Hw In49 m^2 g^4
b117 N^{264-39} w^{ch}/FM4, y^{31d} sc^8 dm B
b118 $N^{264-105}$ (dm)/y Hw In49 m^2 g^4
b119 oc ptg^3·Dp(sc^{S1} y^+)/ClB
b120 oc ptg Tu/sc^{S1} fu In49 sc^8
b121 od
b122 od Dp(f^{+ih}) & y f:=
b123 pa/FM4, y^{31d} sc^8 dm B

b124 peb v
b125 ("bleached") pn w rb cm ct^6 sn^3 ras^2 v dy g^2 f car & y f:=
b126 pn,Inh 1/y Hw in49 m^2 g^4
b127 pn^2
b128 ptg^2
b129 r^{39k} f B/InAM
b130 r^9 & y f:=
b131 ras dy
b132 ras^2
b133 ras^3 m
b134 ras^4 m/ClB
b135 rb
b136 rb cx
b137 rb^{S1}
b138 rg
b139 rst^2/FM1, y^{31d} sc^8 w^a lz^s B
b140 rst^3, In & y f:=
b141 rst^3,In m v ct & y f:=
b142 rst^-(=rst^2)/y Hw In49 m^2 g^4
b143 rux/FM6, y^{31d} sc^8 dm B
b144 rux^2
b145 s
b146 sbr & y f:=
b147 sc
b148 sc cho
b149 sc ct^6 car & y f:=
b150 sc cv v dwx/FM6, y^{31d} sc^8 dm B
b151 sc cv eg
b152 sc cv v f
b153 sc ec cv ct^6 f/ FM3, y^{31d} sc^8 dm B l
b154 sc In49 sn^{x2} car/sc oc ptg sd car
b155 sc In49 v B^{M1}
b156 sc oc ptg sc car/y In49 sn^{x2}·B^S(select B ♀)
b157 sc pn^3 g^2 Bx^2=(g^2 reverted)
b158 sc t^2 v f Tu car & y f:=
b159 sc w BB^L,In·Y^S & y f:=
b160 sc z ec ct^6
b161 sc z w^{17G2} ec ct^6
b162 Sc(Scotched eye)/y sc^{S1} g In49 m sc^8 (select Sc ♀)
b163 scp t
b164 sd f^{ex}/y sc^8 B f In49 v
b165 sd; (se)
b166 shf^2
b167 Sh^2/FM1, y^{31d} sc^8 w^a lz^s B
b168 sn^3
b169 sn^3 lz^{46f24} v & y f:=
b170 sn^3 v B^{M1} & y w f:=
b171 sn^4
b172 sn^{34e}
b173 sn^{36a} & y f:=
b174 sn^c/y In49 m^2 g^4
b175 sp-w
b176 spl
b177 spl rb cx & y f:=
b178 spl rb^{S2}
b179 sta & y f:=
b180 sta/FM3, y^{31d} sc^8 dm B l
b181 su^8-s v
b182 su^2-s w^a cv t
b183 su^3-s cv v f & y f:=
b184 su^{S2}-v-pr v & f B·=
b185 su^{S2}-v-pr v & y f:=
b186 Su^x-dx dx
b187 su-w^a w^a
b188 svr
b189 svr su-w^a w^a
b190 svr w^a
b191 svr^{poi}
b192 sw
b193 sx vb^2 sy/InAM
b194 sy
b195 t
b196 t^2 v f
b197 t^3
b198 Tu & y f:=
b199 tw/FM1, y^{31d} sc^8 sc^8 w^a w^a lz^s B
b200 un Bx^2 & y f:=
b201 un^4
b202 v
v203 v f su^W-f
b204 v f^{3N} car
b205 v r^{12}
b206 v^2 fw
b207 v^{36f}
b208 v^+(rev.v) B^{M2}
b209 v^+(rev.v) B^{M2+}(rev. B, rein.) f^{B15}(mosaic)
b210 v^+(rev.v) f^x Dp(f^{+ih})
b211 vb
b212 vb213 w
b214 w ec
b215 w Om49 lz^s & y f:=
b216 w , f
b217 w^a=apr
b218 w^a f^3 odsy f^{+ih} & y f:=
b219 w^a spl
b220 w^{a2}
b221 w^{a3}
b222 w^{a4}
b223 w^{bf} f^5
b224 w^{bf2}
b225 w^{bl}
b226 w^{Bwx}
b227 w^{ch} wy
b228 w^{co}
b229 w^{co} sn^2
b230 w^{col}
b231 w^{cp}
b232 w^e
b233 w^e sn/ClB
b234 w^{e2}
b235 w^{ec3} (ecru)
b236 w^h
b237 w^i f^3 nn^N
b238 w^{m4}(3C1-2&20)
b239 w^{m4} v w^{mMc}
b240 w^{m4w}
b241 w^{mMc} & y f:=
b242 w^{mMc} f w^{m4}
b243 w^{mR7aH1}
b244 w^{sat}
b245 w^t fw
b246 wy
b247 X^{c1} y & y f:=
b248 X^{c2} cv v f/ClB
b249 X^{c2} ec f & y f:= (ring OK 1957)
b250 X^{c2} y B & y f:= (ring OK 1957)
b251 X^{c2} y f & y w f:= (ring OK 1957)
b252 X^{c2} y v & y f:= (ring OK 1957)
b253 X^{c3} (tm-ac)- sc^8 w^a InS B & y f:=/sc^8·Y (ring from tandem X·X)
b254 y
b255 y ac dvr(+) v bb
b256 (y ac)-51 f (from y f:=) & y f:=
b257 y ac pn In49 v B^{M1}
b258 ("tester-1") y ac pn w rb wy^2 g^2 & y f:=; sc^{19i}/Cy
b259 y ac sc pn & y f:=
b260 y ac sc pn w·Dp(sc^{V1} y^+) & y f:=
b261 y ac sc pn w spl rb cx & y f:=; (sc^{19i}(b pr)/)
b262 y ac sc v & y f:=
b263 y ac v
b264 y ac-53 sc/y Hw In49 m^2 g^4
b265 y ac-53 sc B car·Dp(ac^+ y^+-tm)53 & y f:=
b266 y ac t^2·Dp(sc^{S1} y^+ ac^+) & y f:=
b267 y Aa f:=♀ & B ♂
b268 y B & y f:=
b269 y ct^6 & y f:=
b270 y ct^6 dvr^2 v f & y f:=
b271 y ct^6 f & ac^3 w^a ct f·+
b272 y ct^6 f car & y f:=
b273 y ct^6 f·Dp(y^+ sc^{V1}) & y f:=
b274 y ct^6 t^2 v f car
b275 y ct^{268-42}/FM4, y^{31d} sc^8 dm B
b276 y ct^K

b277 y cv
b278 y f B f^{+ih} & y f:=
b279 y·Dp(y^+ sc^{V1}) & y f:=
b280 y fa wy^2 g^2
b281 y fa^n In49
b282 y fw^{51g} & y f:=
b283 y Hw In49 B^{M1} & y f:=
b284 y Hw In49 m^2 g^4/
w^e sn
b285 y In49 B^{M1} & y f:=
b286 y In49 f car & y f:=
b287 y In49 Fl v g &
y w f:=
b288 y In49 sn^{x2} B^{M1}
& y f:=
b289 y In49 sn^{x2} bb^1 &
y f:=
b290 y In49 v f & y f:=
b291 y In49 v f car
& y f:=
b292 y In49 v ptg oc g^2
& y f:=
b293 y lm1 w In49 f·Y^S/sc^{S1}
B InS
b294 y N^{264-84}/FM6, y^{31d} sc^8
dm B
b295 y oc & y f:=
b296 y pn
b297 y pn w cm ct^6 sn^3 oc
ras^2 v dy g^2 v dy g^2
f od car sw/y sc^{S1} B
In49 v
b298 y pn^{54c} spl
b299 y pn^{54c} w spl
b300 y pn^{54c} w^a
b301 ("tester 3") y rb cn
ras^2 g^2 & y f:=;
sc^{19i}/Cy
b302 y rb cx B^{M1}/y ClB
b303 y rst^3, In car bb
b304 y sc
b305 y sc In49 v g·Dp
(sc^{V1} y^+)
b306 y sc In49 v g f
b307 y sc lz^g v f & y f:=
b308 y sc v g & y f:=
b309 y sl^2 bb^-,In/InAM
b310 y sn oc & y f:=
b311 y sn^3 bb
b312 y t^2 v f
b313 y v
b314 y v & y f:=
b315 y v car bb^-,In/
y InAM
b316 y v f^x B & y w f:=
b317 y v f^x car
b318 y v f^x car su^W-f
b319 y v f^x Dp(f^{+ih}) & y f:=
b320 y w
b321 y w Co & y f:=
b322 y w f
b323 y w f·Dp(sc^{S1} y^+)
b324 y w In49 f
b325 y w sn^3
b326 y w sn^3 f
b327 y w spl
b328 y w t^2 v f
& y f:=
b329 y $w^{-258-11}$ l/y Hw
In49 m^2 g^4
b330 y $w^{-258-11}$ t^2 v
f/y sc^{S1} B InS
b331 y $w^{-258-45}$/y Hw
In49 m^2 g^4
b332 ("doubler") y w^a (l?)·
Dp(B^S)/sc^{S1} In49 v
b333 y^2
b334 y^2 cho^2
b335 y^2 cv y f
b336 y^2 drv^2 v
b337 y^2 ec cv v f car
b338 y^2 In49 lz^s & y f:=
b339 y^2 oc ptg B^{M2} &
y f:=
b340 y^2 oc ptg g,Inh
& y f:=
b341 y^2 sc w^a ec
b342 y^2 su-w^a
b343 y^2 su-w^a w
b344 y^2 su-w^a w^a
b345 y^2 su-w^a w^a spl
b346 y^2 su-w^a w^a spl cv
b347 y^2 su-w^a w^{a2}
b348 y^2 su-w^a w^{a4}
b349 y^2 su-w^a w^{bf}
b350 y^2 su-w^a w^{bl}
b351 y^2 su-w^a w^{co}
b352 y^2 su-w^a w^{col}
b353 y^2 su-w^a w^h
b354 y^2 su-w^a w^{sat}
b355 y^2 su^2-w^a w^a
b356 y^2 v
b357 y^2 v f car
b358 y^2 v f car su-f
& y f:=
b359 y^2 w^a
b360 ("tester-2") y^2 w^a
cm wy^2 g^2 car
& y f:=; sc^{19i}/Cy
b361 y^2 w^a ct f·Dp(y^+
sc^{S1})/ClB
b362 y^2 w^a ct mw f/y
sc^{S1} B InS
b363 y^2 w^a ct^6
b364 y^2 w^a ct^6 lz v f
& y f:=
b365 y^2 w^a cv m f ♂ &
y Aa f:= ♀
b366 y^2 w^a cv v f B
b367 y^2 w^a InS B
b368 y^2 w^a rb^{S1}
b369 y^2 w^a sn^5 B & y
Aa f:=
b370 y^2 w^a spl
b371 y^2 w^a v
b372 y^2 w^a w
b373 y^2 wy^2 g^2 (g^2
Partly reverted?)
b374 y^{2s}
b375 y^{2s} fw^{34e}
b376 y^{3d} & br ec·=
b377 y^{3d} & y f:=
b378 y^{3P},InB
b379 y^4,In cv v f
b380 y^4, In w^a
b381 y^{18cH1}
b382 y^{34c}
b383 y^{bg} ct^6 car
b384 y^{td}
b385 y^{v2}
b386 z w^{11E4}

Scute alleles

(listed alphabetically according to scutes regardless of position of scute in linear order:

c1 sc^2
c2 sc^2 pn & y f:=
c3 sc^{3-1} w & y f:=
c4 sc^{3B}
c5 y sc^4
c6 y sc^4 B f InS &
y f:=
c7 y sc^4 B InS & y f:=
c8 y sc^4 B v^{41b}/y w In49
lz^s
c9 y sc^4 InS w^a; S sc^{19i}
Bl/Cy L^4 sp
c10 sc^5 bb^{sc5}
c11 y sc^5
c12 y sc^5 $w^{-258-48}$ spl;
Dp(1;3)w^{Vco};y f:=
c13 sc^6 car
c14 sc^6 w^a
c15 sc^7
c16 sc^7 InAM car/Df(1)
B^{263-20}
c17 sc^7 oc ptg g,Inh &
y f:=
c18 sc^7 w^a
c19 sc^8
c20 sc^8 B
c21 sc^8 B f^x v & y f:=
c22 (w^r-reddish) sc^8 B InS
w^r & y f:=
c23 sc^8 bb w^a
c24 sc^8 car f In49 v &
y f:=
c25 sc^8 f In49 v & y f:=
c26 sc^8 f v cv & y f:=
c27 (w^r-reddish) sc^8 InS w^r
c28 sc^8 Tu w^a & y f:=
c29 y^{31d} sc^8 w^a
c30 (y ac)B^{270} (dappled) sc^8
B w^a/w In49 lz^s bb
c31 y^{S1} sc^8
c32 y^{S1} sc^8 B f In49 v
c33 y^{S1} sc^8 B f In49 v
w^a & y f:=

c34 y^{S1} sc^8 B In49
c35 y^{S1} sc^8 f InS w^a & y f:=
c36 y^{S1} sc^8 f^3 sd & y w f:=
c37 y^{S1} sc^8 sn^3 w
c38 y^{OX} sc^8 $sn^{5.1}$ w & y f:=
c39 sc^9 Bx f t w^a
c40 sc^{10} w^a
c41 sc^{10-1}/y Hw
c42 sc^{19-}/1J1 sc^{J1}; fes sc^{19i} b pr/Cy dptx^I pr cn^2
c43 sc^{19-}♂ & y f:=; fes sc^{19i} b pr/Cy dptx^I pr cn^2
c44 sc^{28} w^a
c45 sc^{29} w^a
c46 sc^{45} 1/y sc^{S1} B In49 v
c47 sc^{260-14}
c48 sc^{260-22}
c49 sc^C/y sc^{S1} B InS
c50 y sc^{D1}
c51 y sc^{D1}
c52 sc^H,TX^4 & y f:=
c53 1J1 sc^{J1}/Del(sc^7)2 & y f:=
c54 1J1 sc^{J1}/Del(X)24
c55 1J1 sc^{J1}/Del(X^c)Ag (Pontecorvo)
c56 1J1 sc^{S1} car·Dp(ac^+ y^+-tm)53 & y f:=
c57 w^{m5L}; sc^{J4}R♂ & y w f:= (w^{m5L}/)♀
c58 sc^{J6} B & y f:=
c59 sc^{L3},TX^4 (spoon-like)
c60 sc^{L6}
c61 sc^{Mc},TX3/y Hw In49 m^2 g^4
c62 sc^{L8} car m w^a/y w In49 lz^s
c63 sc^{S1} B In49 oc ptg & y f:=
c64 ("plex") sc^{S1} car f In49 v/y ac sc pn w rb cm ct^6 sn^3 ras^2 v dy g^2 f car
c65 sc^{S1} f In49 v w & y f:=
c66 y sc^{S1} B f In49 v & y f:=
c67 y sc^{S1} B In49 sn^{X2} & y f:=
c68 y sc^{S1} B InS & y f:=
c69 sc^{S2},T(1;2)/Cy
c70 sc^{V1},Inp v/y^{S1} sc^8 B f In49 v
c71 sc^{V2}, Inh

Combination of scute or similar inversions

d1 y sc^4 B InS w^a sc^8 & y f:=
d2 y sc^4 In49 sn^{X2} sc^8 & y f:=
d3 y sc^4 B In49 lz^s v sc^{S1}/sc oc ptg sc car
d4 y sc^4 InS sc^{S1} (extra Y in ♀)
d5 y sc^4 InS sc^{S1}/ClB
d6 y sc^8 B InS w^a sc^4 & y f:=; sc^{19i}/Cy cn^2
d7 y sc-(rein. sc^{8-4}) w^a InS B & y f:=; sc^{19i}/Cy lt cn^2
d8 y sc-(rein. sc^{8-4}) w^a InS bb & y f:= sc^{19i}/Cy lt cn^2
d9 y^{S1} sc^8 B InS sc^{S1}/w sn^{5s} bb
d10 y^{S1} sc^8 InS y3P; Cy/Scd
d11 sc-17aH3 f car·sc^{V1}; Cy/sc^{19i}♂ & y f:=; Cy/sc^{19i} ♀
d12 sc^{L8} sc^8 & y f:=
d13 sc^{L8} g^s v lz^g sc^8 & y f:=

sc^{S1}-sc^8 (d14-d45)

d14 sc^{S1} At In49 sc^8
d15 sc^{S1} At In49 v w^a sc^8 & y f:=
d16 sc^{S1} B g In49 m sc^8 & y f:=
d17 sc^{S1} B In49 lz^s sc^8/y ac sc pn w v g f
d18 ("Binsc") sc^{S1} B In49 sc^8 & y f:=
d19 ("Binsn ") sc^{S1} B In49 sn^{X2} sc^8 & y f:=
d20 ("Binsn") sc^{S1} B In49 sn^{X2} sc^8/oc ptg Tu
d21 (Basc") sc^{S1} B InS w^a sc^8
d22 sc^{S1} car B In49 y sc^8 & y f:=
d23 sc^{S1} car m w^a sc^8/w In49 lz^s
d24 sc^{S1} f In49 v w^a sc^8 & y f:=
d25 sc^{S1} In49 m w sc^8/y sn v
d26 ("Insc") sc^{S1} In49 sc^8
d27 ("Insn") sc^{S1} In49 sn^{z2} sc^8 & y f:=
d28 sc^{S1} In49 v sc^8 & sc v f.=
d29 y sc^{S1} At In49 sc^8/oc ptg
d30 ("Binscty") y sc^{S1} B In49 ct^{ns} sc^8
d31 ("Binscy") y sc^{S1} B In49 sc^8 & y f:=
d32 ("new Binscy") y sc^{S1} B In49 sc^8
d33 y sc^{S1} B In49 sc^8/oc ptg (H. Byers' l in sc^{S1} chromosome, c-9-c4)
d34 y sc^{S1} B In49 sn^{X2} v sc^8 & y f:=
d35 y sc^{S1} B In49 sn^{X2} w sc^8 & y f:=
d36 ("Binscty-v") y sc^{S1} B In49 v ct^{ns} sc^8
d37 y sc^{S1} B In49 v sc^8 & y w f:=
d38 y sc^{S1} B In49 v w^a sc^8 & y f:=
d39 ("winscyBx") y sc^{S1} Bx^M In49 w sc^8
d40 y sc^{S1} car odsy f sc^8
d41 y sc^{S1} f In49 v w^a sc^8 & y f:=
d42 ("Inscy") y sc^{S1} In49 sc^8
d43 y sc^{S1} In49 v sc^8
d44 ("winscy") y sc^{S1} In49 w sc^8
d45 y sc^{S1} sc^8
d46 sc^{S1} f InS y^{3P} & y f:=
d47 sc^{V2} B y^{3P}
d48 (y ac)- y^{3P} sc^8 (iso 2952)
d49 (y ac sc)- y^{3P} InS sc^{S1} & y f:=; Cy/sc^{19i}

Translocations of X and 4

e1 TX(1B3+)4 sc^8 B w^a
e2 TX(3C2)4 w^{m5} & y f:=
e3 TX(3C2)4 w^{m5} v f bb/w^{m5} ClB
e4 TX(3C4)4 y $w^{m258-18}$/y Hw In49 m^2 g^4
e5 TX(3C5-6&7)4 N^{8a}/FM6, y^{31d} sc^8 dm B
e6 TX(3E5&6) w^{258-21}/y^{31d} sc^8 B In49 lz^s w^a
e7 TX(4c3)4 & y f:=
e8 TX(9A1)4 & y f:=
29 TX(9B&20)4 "W13"/ClB
e10 TX(9B&20)4 "W13" car
e11 TX(9B&20)4 "W13" sc v^m g/ClB
e12 TX(9B&20)4 "W13" y w & y f:=
e13 TX(11A7)4 & y f:=
e14 TX(13B8-9)4 "Sidly a" & y f:=
e15 TX(16A1)4 B^S & y w f:=
e16 TX(16A1)4 B^S·Y^S/sc·Y^L & y w f:=

Altered Y's sometimes with mutants in X and/or autosomes
(The presence of Y^S and/or Y^L attachments on X·Y chromosomes is uncertain unless they have been freshly tested or are accompanied by markers (bb^+ for Y^S and y^+sc^8 for Y^L) that can be followed.)

f1 y^2 su-w^a w^a Y^S·Y^L y^+ & y v bb·= (no free Y)
f2 Y^S·DpR y X^+ bb·Y^L & y^2 su-w^a w^a bb·= (no free Y)
f3 Y^S·X InEN B y·Y^L & y^2 su-w^a w^a bb·= (no free Y)
f4 Y^S·X InEN B y·Y^L sc^8 y^+ & y^2 su-w^a w^a bb·=; S fes Sp b/(l^+?) InCyL b (no free Y)
f5 Y^S·X InEN In26 B f v·Y^L sc^8 y^+ & y^2 su-w^a w^a bb·= (no free Y)
f6 ("snoc") Y^S·X InEN ptg oc sn^5·Y^L & $\frac{sc\ ct^n\ oc\ ptg\ car}{y\ In49\ sn^{x2}}$)· (no free Y)
f7 ("snoct") Y^S·X InEN ptg oc sn^5·Y^L & $\frac{sc\ ct^n\ oc\ ptg\ car}{y\ ct^1,In\ In49\ sn^{x2}}$)· (no free Y)
f8 Y^S·X InEN v ptg oc sn^5 w y·Y^L sc^8 y^+ & y sc t^2 v f car·= (no free Y)
f9 Y^S·X InEN v y·Y^L(sc^8? y) (no free Y)
f10 Y^S·X InEN v y·Y^L(sc^8? y) & $\frac{sc\ ct^n\ oc\ ptg\ car}{y\ In49\ sn^{x2}}$)·; b pr
f11 Y^S·X InEN v y·Y^L(sc^8? y) & $\frac{sc\ ct^n\ oc\ ptg\ car}{y\ In49\ sn^{x2}}$)·; vg bw
f12 Y^S·X InEN v y·Y^L sc^8 y^+/v; bw^{VA}/L^2 l (no free Y)
f13 Y^S·X InEN y·Y^L sc^8 y^+ (no free Y)
f14 Y^S·X InEN y·Y^L sc^8 y^+ & y^2 su-w^a w^a bb·= (no free Y)
f15 Y^S·X y In49 v f car·Y^L (no free Y)
f16 Y bb^-/w sn bb & y v f·=
f17 Y bb^-/w^{m4w}
f18 Y bb^-/y^2 eq
f19 Y^{st}/w^e bb^1/w^e bb^1/ & bb^1/Y^+;InsNS px sp/lmr^2 (Bridges)
f20 Y:bw^+; net bw crs (iso 1955)
f21 Y:bw^+/y v; bw
f22 Y:bw^+/y v♂ & sc^8·Y/y v; bw (Select)
f23 Y:bw^+/y v♂ & sc^8·Y/y v ♀; S Sp cn bw/dp^{txI} Cy cn bw (Select)
f24 ("MYR") Y^c:bw^+/X^+; bw
f25 ("MYR") Y^c:bw^+?X^{c2} y f; bw (ring OK 1957)
f26 $lJ1^+$·Y/lJ1 sc^{J1} (extra Y in ♀)
f27 ("Maxy") $lJ1^+$·Y/lJ1 $sc^{J1(+)}$ In49 ptg oc B^{M1}/y sc^{S1} car odsy f g^2 dy v ras^2 sn^3 ct^6 cm rb ec w pn l sc^8
f28 ("Maxy-v") $lJ1^+$·Y/lJ1 $sc^{J1(+)}$ In49 v ptg oc B^{M1}/y sc^{S1} car odsy f g^2 dy v ras^2 sn^3 ct^6 cm rb ec w pn l sc^8
f29 sc^8·Y/ac^3
f30 sc^8·Y/In(X^{c2})w^{vc} f & y f:=
f31 sc^8·Y/In49 ptg oc B^{M1} & y f:=
f32 sc^8·Y/lJ1 sc^{J1} & y f:=
f33 sc^8·Y/lJ1 sc^{J1} In49 v B^{M1} & y f:=
f34 sc^8·Y/l (y ac)$^-$ B In49 sn^{x2} sc^8 & y f:= (from X-r. oogonia $24)
f35 ("Max-Tu") sc^8·Y/l (y ac)$^-$ Tu B In49 sn^{x2} sc^8/y ac pn w rb cm sn^3 ct^6 oc ras^2 v dy g^2 f od car sw
f36 sc^8·Y/oc ptg & y f:= (iso 1956)
f37 sc^8·Y/sc w B·Y^S & y f:=; Cy,In/S Sp ab^2 ltd
f38 sc^8·Y/sc w ct f·Y^S & y f:=; Cy,In/S Sp ab^2 ltd
f39 sc^8·Y/sc^{V1} v & sc^8·Y/y f:= ; sc^{19i}/Cy lt^3
f40 sc^8·Y/Tam(X;3) & sc^8·Y/y f:=
f41 sc^8·Y/X^{c2} y f & y f:= (ring OK 1957)
f42 sc^8·Y/X^{c2} y v & y f:= (ring OK 1957)
f43 sc^8·Y/y ac sc B·Dp(sc^{S1} ac^+ y^+) & y N^W f:= (N with w^+
f44 sc^8·Y/y ac sc oc ptg & y f:=
f45 sc^8·Y/y ac^{-53} sc & y f:=
f46 sc^8·Y/y B♂ & y f:= ♀(to cross ♂ by y sc^{S1} In49 sc^8; bw; st p^p ♀)
f47 ("multi-♂") sc^8·Y/y In49 B & y f:=; bw^D
f48 sc^8·Y/y In49 B^{M1}
f49 sc^8·Y/y In49 v F1 g & y f:=
f50 sc^8·Y/y In49 v F1 g & y In49 v F1 g/pn,Inh
f51 sc^8·Y/y In49 v f
f52 sc^8·Y/y In49 v f B·Y^L & y f:=
f53 sc^8·Y/y sc w In49 v g f

f54 sc^8·Y/y sc w In49 v g f & y f:=
f55 sc^8·Y/y sc^4 B f InS & y f:=
f56 sc^8·Y/y sc^4 B f InS w^a & y f:=
f57 sc^8·Y/y sc^4 B InS & y f:=
f58 sc^8·Y/y sc^4 B InS w^r sc^8 & y f:=
f59 sc^8·Y/y sc^4 f InS w^a & y f:=
f60 sc^8·Y/y sc^4 w sc^8 (sc^8·Y in ♂ & ♀)
f61 ("Multipare D") sc^8·Y/y sc^{S1} B InS/y Hw In49 m^2 g^4; (ci gvl ey^R sv^n)
f62 sc^8·Y/y sc^- (rein. sc^{8-4}) B·Dp(sc^{S1} ac^+ y^+) & y f:=
f63 sc^8·Y/y v & y f:=
f64 sc^8·Y/y w^{m4}
f65 sc^8·Y/y^2 w^a sn^5 B & y N^M f:= (N with w)
f66 sc^8·Y/y^{S1} sc^8 B f In49 v
f67 sc^8·Y·B^S/lJ1 y & y ct^6 f·=
f68 sc^8·Y·B^S/sc^8 B In49 w & y f:=; (ho ed cl/+)
f69 sc^8·Y·B^S/y w^{m4} ras^2
f70 sc^8·Y·B^S/y w^{m4}; dp
f71 sc^8·Y·B^S/y w^{m4}; (ho) ed cl
f72 sc^8·Y·B^S/y^2 ct^6 & y f:=
f73 sc^8·Y·B^S/y^2 sd^2 & y f:=
f74 sc^8·Y·B^S/y^2 w^i & y f:=
f75 sc^8·Y·B^S/y^2 w^i ct^6 & y f:=
f76 sc^8·Y·B^S/y^2 w^i ct^6 f & y f:=
f77 sc^8·Y:bw^+/ac^3; bw
f78 sc^8·Y:bw^+/ac^3; cn bw
f79 sc^8·Y:bw^+/sc^8 B In49 w; bw
f80 sc^8·Y·w^+/y w^a
f81 Y^L/f·Y^S & sc v f·=
f82 Y^L/f·Y^S & y^2 wy^2 g^2 f·=
f83 Y^{Lc}/InEN2·Y^S & y ct^6 f·= (InEN2 from X^{c2} opened)
f84 ("Y^{Lc} snocty") Y/oc ptg·Y^S & Y^{Lc}/y ct^n oc ptg car / y ct^1,In In49 sn^{x2})·
f85 Y^{Lc}/oc ptg·Y^S & y v f·=; S Sp ab^2 ltd/Cy,Ins cn^2
f86 ("Y^{Lc} snocty; bw") Y^{Lc}/oc ptg·Y^S & Y^{Lc}/y ct^n oc ptg car / y ct^1,In In49 sn^{x2})· ;bw
f87 Y^{Lc}/w oc ptg·Y^S & Y^{Lc}/y^2 X^+·w^a InS B sc^8 (tandem X·X giving rings)

Sterilizer ("sz") stocks (f88-f97)

f88 ("sz w") Y^{Lc}/w·Y^S
f89 ("sz +") Y^{Lc}/X·Y^S
f90 ("sz bw") Y^{Lc}/X·Y^S; bw
f91 ("sz bw e") Y^{Lc}/X·Y^S; bw; e
f92 ("sz c") Y^{Lc}/X·Y^S & y v f·=; c
f93 ("sz e") Y^{Lc}/X·Y^S & y v f·=; e
f94 ("sz lz f") Y^{Lc}/lz^3 f·Y^S & y v f·=
f95 ("sz lz m f") Y^{Lc}/lz^3 m f·Y^S & y v f·=
f96 ("sz m f") Y^{Lc}/m f·Y^S & y v f·=
f97 ("sz y w") Y^{Lc}/y w·Y^S & y ct^6 f·=
f98 Y^{Lc}/y v·Y^S; bw^{VA}/L^2 l
f99 Y^{Lc}/y v B·Y^S & ac^3 w^a ct^6 f·=
f100 (new "facl", 1959) Y^{Lc}/y sn oc ptg·Y^{Lc}/y^2 oc ptg B^{M1}/sc^{S1} In49 sn^{x2} sc^8
f101 ("jynd") Y^{Lc}/y sn^5 oc ptg v·Y^S ♂ & Y^{Lc}/sc^{J1} pn w rb cm ct^6 oc ras^2 v dy g^2 f od car sw/y sc^{S1} B In49 sn^{x2} sc^8 ♀
f102 Y^{Lc}/y In49 v f·Y^S & y sc t^2 v f car·=
f103 Y^{Lc}/y w sn^5 oc ptg·Y^S & y v f·=
f104 Y^{Lc}/y^2 oc ptg fu·Y^S & Y^{Lc}/y w^a·=
f105 sc·Y^L/oc ptg·Y^S & sc·Y^L/y f:=; Cy,Ins cn^2/S Sp ab^2 ltd
f106 sc·Y^L/sc w B·Y^S & y f:=; Cy,In/S Sp ab^2 ltd
f107 sc·Y^L/sc w BB^L,In·Y^S & y f:=
f108 sc·Y^L/sc w ct^6 f·Y^S & y f:=; Cy,In/S Sp ab^2 ltd
f109 sc·Y^L/y ac sc ct^6 f·Y^S
f110 sc·Y^L/y In49 v f·Y^S

f111 sc·Y^L/y In49 v f·Y^S;e
f112 sc·Y^L/y sc$^-$(rein.sc$^{8-4}$)·Y^S & y f:=
f113 sc·Y^L/y·Y^S & y f:=; cn bw; (e)
f114 sc·Y^L/y^2 v f·Y^S & y w f·=
f115 sc·Y^L/y^2 w^a ct^6 f·Y^S ♂ & sc·Y^L/ y^2 X^+·sc^8 w^a InS B ♀ (tandem X·X giving rings)
f116 y^3·Y^L/sc w oc f·Y^S & y f:=
f117 y^3·Y^L/sc^{V1-} oc lz^3·Y^S & y f:=
f118 y^3·Y^L/sc^{V1-} w·Y^S & y f:=
f119 y^3·Y^L/y ct^6 oc lz·Y^S & y f:=
f120 ("plond") y^3·Y^L/y^2 oc lz·Y^S ♂ & y^3·Y^L/ y ac sc pn w rb cm ct^6 sn^3 oc ras v m g^2 f car/sc^{S1} B In49 lz^s sc^8 ♀
f121 Y^S/g^2 B·Y^L & y f:= (Stern) (dp^I)
f122 Y^S/y ct^6 f·Y^L & y f:=
f123 Y^S·Y^S#2/y v f·Y^L & y f:=
f124 sc^{V1}·Y^S/y In49 v·Y^L & y f:=
f125 sc^{V1}·Y^S/y In49 v B·Y^L & y f:=
f126 sc^{V1}·Y^S/y In49 v f B·Y^L & y f:=
f127 sc^{V1}·Y^S/y In49 v ptg oc f B·Y^L & y f:=
f128 sc^{V1}·Y^S/y v f bb^+·Y^L & y f:=
f129 TY2G/b pr (tk)
f130 TH3(II4Aa^3) l/ru h D InsCXF ca (TY3 in and)
f131 Tp4:Y (2 Y:4's in both ♂ & ♀) (Transpos. Edmondson)
f132 Tp4:Y/Basc & y·=; ci ey^R
f133 Tp4:Y/Cat/Cat ♂ & Cat/M4 ♀ (unselected)
f134 Tp4:Y/ci^D
f135 Tp4:Y/ey^D or Cat ♂ & ey^D/Cat ♀
f136 Tp4:Y/X^+ & y f:=

Chromosome 2*

*S^2 and/or Cy are to be understood always to be accompanied by InCyL and cn^2 by InCyR even where not so designated. When cn is present InCyR is absent. Ins following S^2 or Cy after a comma refers to both of these inversions, but InL only to the left-arm one. If either of these inversions is designated in a chromosome without the other, the latter should be understood to be absent. InMis designates the long pericentric inversion of Mislove.

g1 a px or
g2 a px sp
g3 ab
g4 ab^2/S^2 Ins(CyL,CyR) lc cn^2
g5 ab^2/T(Y;2)E
g6 ab^2 cn^4 Pm^1/Cy pr Bl cn^2 L^4 sp^2
g7 ab^2 ix^2 bw sp^2/Cy, dp^{Th} Bl L^4 sp^2
g8 ab^2 InCyR L^4 sp^2/b InsNSL&R mr
g9 ab^2 ms ta crs/Cy pr Bl cn^2 L^4 sp^2
g10 abr/SM5, al^2 Cy lt^V sp^2
g11 ad
g12 al
g13 al b c sp
g14 al b cn sp (iso)
g15 al dp b bw l(2)ax/SM5, al^2 Cy lt^V sp^2
g16 al dp b pr
g17 al dp b pr blt bw/SM5, al^2 Cy lt^V sp^2
g18 ("apl") al dp b pr c px sp
g19 ("twelvepl") al dp b pr cn vg c a px bw mr sp/S^2 Cy lt^3 pr^+ Bl cn^2 L^4 sp^2
g20 al S ast ho/Cy, En-S
g21 al^2 Cy ab^{51g} pr Bl cn^2 L^4 sp^2/S Sp cn bw sp
g22 al^2 Cy,InL lt^3/b pr Bl lt^3 cn^2 InCyR L^4 sp^2
g23 al^2 Cy pr Bl cn^2 InCyR c vg sp^2/InsNS px sp
g24 al^2 Cy pr Bl cn^2 L^4 bw sp^2/InsNS px sp
g25 al^2 InMis dp^{txI} Cy cn^2 L^4 sp^2/S Sp U, InLR bw
g26 al^2 InMis dp^{txI} Cy pr Bl cn^2 L^4 sp^2/ S Sp U,InLR bw
g27 Alu
g28 an/SM5, al^2 Cy lt^V sp^2
g29 ang
g30 ant; (ro)
g31 ap^4/Rvd,In2LR
g32 ap^4/SM5, al^2 Cy lt^V sp^2
g33 arch ch1/SM5, al^2 Cy lt^V sp^2
g34 ast ho
g35 ast ho ed dp cl
g36 ast^4 dp cl
g37 ast^4 dp cl sp
g38 b
g39 b cn bw
g40 b el rd^s pr cn
g41 b gp
g42 b Go/Gla
g43 b j
g44 b l(2)Bld pr c px sp/SM5, al^2 Cy lt^V sp^2
g45 b lt bw
g46 b lt l cn mi sp/b In(2)bw^{VDel}
g47 b lt wxt bw
g48 b nub pr
g49 b pr
g50 b pr Bl tk/S^2 Cy cn^2 L^4 sp^2
g51 b pr c px sp
g52 b pr tk
g53 b pr tk/T(Y;2)G
g54 b sf
g55 b vg
g56 Bl/esc
g57 Bl/Cy, bw^{45a} sp^2 or^{45a}
g58 Bl/In(2LR)dp
g59 Bl $bw^{"VA"}$T(2;3)/Cy,In^L L^2
g60 Bl L^2/Cy, dp^2
g61 Bl stw^3/In(2LR)dp
g62 Bl stw^{48} blt tuf/SM5, al^2 Cy lt^V sp^2
g63 Bla/SM5, al^2 Cy lt^V sp^2
g64 blo
g65 blt
g66 bran
g67 bri
g68 bs^2
g69 bw (iso 2, 1959)
g70 bw ba
g71 bw sp (iso 1954)

g72 bw^D
g73 bw^{2b}
g74 bw^4
g75 bw^5-/Cy cn^2 L^4 sp^2
g76 bw^{-5}/Cy, dp^2
g77 c
g78 c bw
g79 c px bw^D sp
g80 c wt px
g81 cg c/U,InLR
g82 ch
g83 chl
g84 chl en/SM5, al^2 Cy lt^V sp^2
g85 chl l(2)bw bw^{2b} mr^2/SM5, al^2 Cy lt^V sp^2
g86 chy
g87 ck/SM5, al^2 Cy lt^V sp^2
g88 cl
g89 cl^2 px/T(Y;2)E
g90 cn (iso 2)
g91 cn bw
g92 cn bw sp
g93 cn px,InLR crs/S^2 dp^{txi} Cy pr Bl cn^2 L^4 sp^2
g94 cn sp (iso 1954)
g95 cn^2 InCyR cg sp^2/InsNS px sp
g96 cn^3 cg bw^5- mr/Cy cn^2 L^4 sp^2
g97 cn^3/T(Y;2)C
g98 cn^{35k}
g99 cg
g100 cr-u/Cy; (w^e)
g101 d/SM5, al^2 Cy lt^V sp^2
g102 da/SM1, al^2 Cy sp^2
g103 Df(2)42, en/Cy, al^2 lt^3 L^4 sp^2
g104 Df(2)al/Cy, En-S
g105 Df(2)$bw^{VDe2}L_{CyR}$/Gla
g106 Df(2)MB/SM1, al^2 Cy sp^2
g107 Df(2)MS4/SM1, al^2 Cy sp^2
g108 Df(2)MS8/Cy, al^2 lt^3 Dp(2;2)41 L^4 sp^2
g109 Df(2)MS8/SM1, al^2 Cy sp^2
g110 Df(2)MS10/Cy pr, $Dp(2;2)41^2$
g111 Df(2)MS10/SM1, al^2 Cy sp^2
g112 Df(2)Px/Df(2)P;Dp(2;3)P/In(3R)Mo, sr; w^e
g113 $Df(2)rl^{10a}$ lt cn/Cy
g114 $Df(2)rl^{10b}$ lt cn/Cy, al^2 lt^3 L^4 sp^2
g115 Df(2)S2/Cy, En-S
g116 Df(2)S3;Dp(2;2)a, Cy, En-S
g117 Dke c
g118 dil^2 hv bw sp/SM5, al^2 Cy lt^V sp^2
g119 dp
g120 dp b cn sp/al^2 Cy pr Bl cn^2 L^4 sp^2
g121 dp b L^4 Pm^1/IndpT23 b
g122 dp bw^5- mr/al^2 InMis Cy cn^2 sp^2
g123 dp cn bw
g124 dp^o
g125 dp^{o2}
g126 dp^{o3} ta sp/Cy pr Bl cn^2 L^4 sp^2 (iso 2)
g127 dp^{Nov}
g128 dp^{Rf}/SM5, al^2 Cy lt^V sp^2
g129 dp^T ab^2 pr Bl rnT23 InNSR mr/al^2 Cy cn^2 L^4 sp^2
g130 sp^T Sp ab^2 cn bw sp/S^2 ls Cy pr InsL&R cn^2 bw sp
g131 dpT Sp cn bw sp/S^2 (ls^+) Cy,InL cn bw sp
g132 dp^T Sp cn InRSR mr/S^2 ls Cy pr Bl cn^2 L^4 bw sp^2
g133 dp^{tx} b/Cy,Ins cn^2
g134 dp^{tx} b/SM5, al^2 Cy lt^V sp^2
g135 dp^{tx} Sp ab^2/S^2 ls Cy,InCyL
g136 dp^{tx} Sp b/S^2 ls, InCyL
g137 dp^{tx} Sp b/S^2 ls, InCyL b
g138 dp^{tx} Sp cn/S^2 Cy,InCyL cn
g139 dp^{tx} Sp cn bw/S^2 Cy,InCyL cn bw
g140 dp^{tx} Sp cn^2/S^2 Cy cn^2 (homoz. InCyR)
g141 dp^{txI} Cy Bl cn^2 L^4 sp^2/InNSL InNSR px sp
g142 dp^{txI} Cy,Ins04 pr cn^2/InsNS px sp
g143 dp^{txI} Cy,Ins06 pr cn^2/InsNS px sp
g144 dp^v; vo-3 (2;3)
g145 dp^{v2}
g146 dp^{v1}/SM5, al^2 Cy lt^V sp^2
g147 ds dp
g148 ds ft dp^{v2} l(2)M b pr/SM5, al^2 Cy lt^V sp^2
g149 ds S G b pr/Cy, al^2 lt^3 L^4 sp^2
g150 ds^{38k}/Cy(2L), dp^2 b pr
g151 ds^W/In(2L)Cy-t, Su-S sp^2 pr
g152 dsr
g153 dw-24F cl/SM5, al^2 Cy lt^V sp^2
g154 ed dp cl
g155 ed Su^2-dx
g156 el
g157 ex
g158 ex ds S^X ast^X/SM1, al^2 Cy sp^2
g159 fes Alu lt/SM5, al^2 Cy lt^V sp^2
g160 fes dp^{tx} Sp/al^2 Cy lt^3 (L^4) sp^2
g161 fes IndpT23 b sp/al^2 Cy cn^3 L^4 sp^2
g162 fes ms cn sp/net dp^{txI} Cy b pr Bl lt^3 cn^2 L^4 sp^2 (iso 1957)
g163 fes pr rnT23/al^2 Cy b cn^2 L^4 sp^2
g164 fj l(2)Su-H/SM5, al^2 Cy lt^V sp^2
g165 fj wt/SM5, al^2 Cy lt^V sp^2
g166 fr/Cy, sp^2
g167 fr^2 wt/SM5, al^2 Cy lt^V sp^2
g168 ft
g169 Grv/SM5, al^2 Cy lt^V sp^2
g170 Gla,InLR/S^2 Cy cn^2 bw sp
g171 hk
g172 hk pr
g173 ho
g174 hv/SM5, al^2 Cy lt^V sp^2
g175 Hx
g176 hy/SM5, al^2 Cy lt^V sp^2
g177 In(2)bw^{VDe1}/b lt l cn mi sp
g178 In(2)bw^{De2}/Rev.l
g179 In(2L)Cy, al^2 ast^3 b pr (Cy not present)
g180 In(2L)Cy, b pr cn^2 In(2R)Cy
g181 In(2L)t, esc c sp/SM5, al^2 Cy lt^V sp^2
g182 In(2L)t, lt l L^4 sp^2/ds^{33k} Pm
g183 Ins(2L+2R)Cy, al^2 En-S sp^2 (homozygous)
g184 In(2LR)102, ds^W/SM1, al^2 Cy sp^2

g185 InNSL InNSR/al^2 Cy,InL lt^3 L^2
g186 j
g187 j-1 ab^2 InNSR mr/S^2 dp^{txi} Cy cn^2 bw sp
g188 J Bl/In(2L)NS
g189 J^{34e}
g190 kn
g191 l(2)39a px slt sp/SM5, al^2 Cy lt^V sp^2
g192 l(2)a bs^3, In(2L)t/ds^{33k} Pm
g193 l(2)ay b c sp/SM5, al^2 Cy lt^V sp^2
g194 l(2)gl cn bs/SM5, al^2 Cy lt^V sp^2
g195 l(2)H L^2/SM5, al^2 Cy lt^V sp^2
g196 l(2)mat/SM5, al^2 Cy lt^V sp^2
g197 l(2)me/SM1,al^2 Cy sp^2
g198 L
g199 L^2
g200 L^4
g201 L^5
g202 L^G
g203 L^K
g204 L^r
g205 L^{si}
g206 ll^2
g207 lm/Cy, S^2 dp^2 En-S
g208 ls dp^1/al^2 Cy cn^2 L^4 sp^2
g209 ls dp^T Sp ms ta cn crs/S^2 Cy lt^3 pr$^+$ Bl cn^2 L^4 sp^2
g210 lt/T(Y;2)A
g211 lt bw
g212 lt bw^{Amherst}
g213 lt std/SM1, al^2 Cy sp^2
g214 lt stw^3
g215 lt^3 Dp(2;2)41 L^4 sp^2 In(2R)Cy/ds^{33k} Pm
g216 ltd
g217 lw
g218 lys rc; ss (2;3)
g219 M(2)33a/al^2 InMis Cy cn^2 sp^2
g220 M(2)173/SM5, al^2 Cy lt^V sp^2
g221 M(2)B/In(2L)t, l(2)B
g222 M(2)B/SM5, al^2 Cy lt^V sp^2
g223 M(2)l^2/ds^{33k} Pm
g224 M(2)l^2/SM1, al^2 Cy sp^2
g225 M(2)p/Cy, al^2 lt^3 L^4 sp^2
g226 M(2)S3/SM1, al^2 Cy sp^2
g227 M(2)S6/SM5, al^2 Cy lt^V sp^2
g228 M(2)S7/SM5, al^2 Cy lt^V sp^2
g229 M(2)S9/SM5, al^2 Cy lt^V sp^2
g230 M(2)S11/Cy, bw^{V34}
g231 M(2)S11/SM5, al^2 Cy lt^V sp^2
g232 M(2)z/SM5, al^2 Cy lt^V sp^2
g233 M(2)z Sk b/Cy(2L)dp^2 b pr
g234 mi/Pm^2
g235 mn/Cy cn^2 L^4 sp^2
g236 mr bs^2/Cy, sp^2
g237 mr bs^2/ds^{33k} Pm
g238 mr^2/Bld, In(2R)Cy
g239 ms bw/Cy pr Bl cn^2 L^4 sp^2 (iso 1955)
g240 ms cn bw/dp^{txI} Cy pr Bl lt^3 cn^2 L^4 sp^2
g241 ms cn rm/Cy cn^2 L^4 sp^2
g242 ms cn sp/dp^{txI} Cy pr Bl lt^3 cn^2 L^4 sp^2
g243 msf/SM5, al^2 Cy lt^V sp^2
g244 net
g245 net al ex ds S ast shv ho rub/SM1, al^2 Cy sp^2
g246 net b cn crs/dp^{txI} Cy pr Bl lt^3 cn^2 L^4 sp^2 (iso 1955)
g247 net bw crs/dp^{txI} Cy pr Bl lt^3 cn^2 L^4 sp^2 (iso 1955)
g248 net bw mr crs/al^2, InMis di^{txI} Cy Bl cn^2 L^4 sp^2 (iso 1956)
g249 net bw sp
g250 net dp b pr cn/dp^{txI} Cy pr Bl cn^2 L^4 sp^2
g251 net ed Su^2-dx
g252 net ta sp/al^2 ly pr Bl cn^2 L^4 vg sp^2
g253 net ta vg^{S2} sp/dp^{txI} Cy pr Bl cn^2 L^4 sp
g254 nub^2
g255 nw^2/Cy-RNS
g256 pd
g257 pd ll^2 sp
g258 Pfd/SM5, al^2 Cy lt^V sp^2
g259 pi/SM5, al^2 Cy lt^V sp^2
g260 pi l(2)301/SM5, al^2 Cy lt^V sp^2
g261 Pin
g262 pk cn
g263 Pm^1/T(Y;2)G
g264 Pm^2/mi sp^2
g265 po vg
g266 po^2
g267 pr
g268 pr cn/T(Y;2)C
g269 pr cn ix/SM5, al^2 Cy lt^V sp^2
g270 pr^{bw}
g271 pu
g272 puf
g273 pw-c/SM5, al^2 Cy lt^V sp^2
g274 px
g275 px bl (old Berlin stock of Goldschmidt) bl=bs?
g276 px bw mr sp/ds^{33k} Pm
g277 px bw sp/T(Y;2)J
g278 px slt sp
g279 Px^2-/Cy cn^2 L^4 sp^2
g280 Px^{-2}, bw sp/SM1, al^2 Cy sp^2
g281 pys
g282 Q
g283 rd/SM5, al^2 Cy lt^V sp^2
g284 rdo
g285 rdo^2 pr
g286 rh
g287 rk cn bw (iso 2)
g288 rl
g289 rnT23/Cy Bl cn^2 L^4 sp^2
g290 rub
g291 Ruf/ds^{33k} Pm

g292 S/Cy, En-S
g293 S dp^1/al^2 Cy cn^2 L^4 sp^2
g294 S fes Alu lt/al^2 Cy cn^2 L^4 sp^2
g295 S fes Sp b/Cy b lt^3 cn^2 L^4 sp^2
g296 S fes Sp ms ta cn mr crs/al^2 InMis dp^{txI} Cy pr Bl cn^2 L^4 sp^2
g297 S Sp ab^2 ap^4 InNSR px sp/al^2 Cy Bl cn^2 L^4 sp^2
g298 S Sp Bl bw^D/Cy cn^2 lc
g299 S Sp Bl L^2 Px^-/dp^{txI} Cy,InsO pr cn^2
g300 S Sp Bl L^{rm} bw^D/dp^{txI} Cy,InsO pr cn^2
g301 S Sp Bl Pfd Bw^D/dp^{txI} Cy,InsO pr cn^2
g302 S Sp cn bw/dp^{txI} Cy cn bw
g303 S Sp crs/al^2 Cy pr Bl L^4 sp^2
g304 S Sp InNSR mr/dp^{txI} Cy pr Bl cn^2 L^4 sp^2
g305 S Sp (ls?) cn/dp^{txI} Cy cn
g306 S Sp (ls^+?) cn bw sp/dp^{txI} Cy,InL cn bw sp
g307 S Sp ms ta cn crs/al^2 InMis dp^{txI} Cy pr Bl cn^2 L^4 sp^2
g308 S Sp ms ta cn crs/dp^{txI} Cy,InsO pr cn^2
g309 S Sp pr Bl rnT23 InNSR mr/dp^{txI} Cy pr cn^2
g310 S Sp pr cn^2 InCyR/dp^{txI} Cy pr cn^2
g311 S Sp rnT23/dp^{txI} Cy pr Bl cn^2 L^4 sp^2
g312 S^2 ab^{51b} InCyL/dp b L^4 Pm^1
g313 S^2 Cy lt^3 pr^+ Bl cn^2 L^4 sp^2/InNSL InNSR px sp
g314 S^2 dp^{txI}, InCyL/ls Sp b
g315 S^R/ds^{33k} Pm
g316 sca
g317 sca l(2)C/SM5, al^2 Cy lt^V sp^2
g318 sf^2
g319 shr bw^{2b} abb sp/SM5, al^2 Cy lt^V sp^2
g320 shv
g321 shv ho
g322 sn px/SM5, al^2 Cy lt^V sp^2
g323 sm px pd/SM5, al^2 Cy lt^V sp^2
g324 so
g325 so^2 b cn
g326 sp^2 bs^2
g327 Sp/In(2L)t, l(2)R
g328 Sp/Sm5, al^2 Cy lt^V sp^2
g329 Sp pur cn InNSR px sp/Cy pr Bl cn^2 L^4 bw sp
g330 Sp J/In(2L)Cy-t, Su-S dp^2 pr
g331 Sp J/SM5, al^2 Cy lt^V sp^2
g332 Sp J L^2 Pin/SM5, al^2 Cy lt^V sp^2
g333 Sp ms cn mr crs/Cy pr Bl cn^2 L^4 sp^2
g334 spd gt-4/Gla,InLR
g335 sple
g336 spt
g337 std/SM5, al^2 Cy lt^V sp^2
g338 stw
g339 stw^2
g340 stw^3
g341 stw^3/T(Y;2)B
g342 stw^5
g343 stw^{48} blt tuf
g344 ta cn bw/al^2 Cy pr Bl cn^2 L^4 sp^2
g345 ta cn bw sp/Cy pr Bl cn^2 L^4 sp^2
g346 Tft/Cy
g347 tkd/SM5, al^2 Cy lt^V sp^2
g348 tkv
g349 tri vg^{No2}/SM5, al^2 Cy lt^V sp^2
g350 tuf ltd
g351 U/cg c
g352 Uf
g353 vg (iso 2,3)
g354 vg bw
g355 vg^B/SM5, al^2 Cy lt^V sp^2
g356 vg^C/Rvd,In2LR
g357 vgC/SM5, al^2 Cy lt^V sp^2
g358 vg^{-D}/SM5, al^2 Cy lt^V sp^2
g359 vg^{-D} sp^2/Cy cn^2 L^4 sp^2
g360 vg^{ni}
g361 vg^{np}
g362 vg^{nw} Hia/SM5, al^2 Cy lt^V sp^2
g363 vg^{nw} Hia/$T(2;3)S^M$ Cy
g364 vg^U/Roi, bw sp or
g365 vst/SM5, al^2 Cy lt^V sp^2
g366 whd
g367 wt

Chromosome 3
(containing genes of 2 in a few cases)

h1 a-3
h2 aa h
h3 abd
h4 app
h5 as^{hg}
h6 bar-3(Ives)
h7 Bd^G/In(3R)C, l(3)a
h8 bf/In(3R)C, Sb e l(3)e
h9 bp/TM1, Me ri
h10 bul
h11 bv
h12 bx^3 Cbx Ubx bxd pbx/Xa
h13 bx^{34e}
h14 bx^D=Ubx
h15 ca
h16 ca bv
h17 ca K-pn
h18 ca^2
h19 ca^{572} $jIIIa^3$/Me,Ins ri Sb^1
h20 Cbx
h21 cd
h22 cmp ca/In(3R)C, e
h23 Cor/ru h D InsCXF
h24 cp in ri p^p
h25 cp
h26 cu
h27 cu kar
h28 cur
h29 cv-c
h30 cv-c sbd^2
h31 cv-d
h32 D/Gl
h33 D InsCXF/Tri
h34 D tra/InLP Dfd InRP ca
h35 D^3 H/InsP
h36 D^3 Sb ca^2/Payne
h37 det
h38 Df(3)MS31/T(2;3)Me
h39 Df(3)sbd^{105}/Xa
h40 Dfd/In(3LR)Cx
h41 Dfd^r
h42 Dl H e^s cd/In(3R)spr, spr
h43 Dl^3In(3R)C, e
h44 Dl^5/In(3R)C, l(3)a
h45 Dl^{14}/In(3R)Cyd, Cyd
h46 Dl^x/Payne

h47 drb
h48 dwh/Payne, Dfd ca
h49 e P^{1}/ru h D InsCXF e
h50 e^{4} wo ro
h51 e^{11}
h52 e^{s}
h53 eg/In(3LR)Cx
h54 eg^{2}/In(3LR)Cx
h55 eyg
h56 fz
h57 gl
h58 gl^{2} e^{4}
h59 gl^{3}
h60 Gl bx^{D}/InsLVM
h61 Gl Sb H/Payne
h62 gs
h63 h
h64 h ri
h65 h ri ca (iso 1953)
h66 h ri e^{s} (iso 1957)
h67 h^{2}
h68 H/In(3R)hp, hp
h69 H Pr/In(3R)C, e
h70 H^{2}/Xa
h71 H^{3}/In(3R)C, Sb e l(3)e
h72 Hn^{r} h ri/ru h D Sb InsCXF
h73 Hn^{r3} sr
h74 in
h75 In(3L)$p^{mot-36e}$/R
h76 In(3L)P, In(3R)P18, Me Ubx e^{4}/In(3LR)Cx
h77 Ins(3)Ubx^{130}/T(2;3)Xa
h78 In(3R)$Antp^{B}$?TM1, Me ri
h79 In(3R)Dl^{B}, st Dl^{B}/In(3R) P^{W}, st l(3) W ca
h80 In(3R)Hu, Hu Sb^{Spi}/Payne
h81 In(3R)M0, sr/Xa, ca
h82 In(3R)P^{FLA} (homozygous)
h83 jv
h84 jv Hn^{r} h
h85 jvl
h86 kar^{2}
h87 Ki
h88 l(3)ac e^{s} M(3)w/LVM
h89 l(3)36d10/In(3LR)Cx, D
h90 l(3)tr Sb/In(3LR)Ubx^{130}, Ubx^{130} e^{s}
h91 l(3)tr Ubx/TM1, Me ri Sb^{1}
h92 ld
h93 Ly/D3
h94 Ly Sb/LVM
h95 M(3)1/In(3R)C, l(3)e
h96 M(3)36e/In(3R)C, l(3)a
h97 M(3)40130/Payne, Dfd ca
h98 M(3)B/In(3R)C, e l(3)e
h99 $M(3)B^{2}$/In(3R)C, Sb e l(3)e
h100 M(3)S32/T(2;3)Me
h101 M(3)S34/T(2;3)Me
h102 M(3)S36/T(2;3)Me
h103 M(3)S37/Me
h104 M(3)w/In(3R)C, e l(3)e
h105 M(3)y/Me
h106 ma
h107 ma fl
h108 mah
h109 Mc/Xa
h110 Me,InL bx^{D}/ru h D InsCXF Sb
h111 Me,InL InRC e l3e/ru h D InsCXF Sb e^{s}
h112 Me,InL Sb/ru h D InsCXF
h113 Me,Ins ri Sb^{1}/ru h D InsCXF ca
h114 Me,Ins ri Sb^{1}/D^{3} st ri InRC e l3e
h115 Mio
h116 N-X/Xa
h117 obt
h118 p
h119 p^{p}
h120 p^{p} bx sr e^{s}
h121 p^{p} cu
h122 pb/In(3LR)Cx
h123 pbx/Xa
h124 Pc/TM1, Me ri
h125 Pr/In(3R)C, e
h126 Pr Dr/TM3, y^{+} ac^{+} ri p^{p} sep bx^{34e} e^{s}
h127 Pr^{K} Dp/InPL InPR (Krivshenko)
h128 Pt/Xa, ca
h129 pyd
h130 R Ly/In(3L)P, gm
h131 ra
h132 red (Malpighians)
h133 red (Malpighians) e
h134 ri
h135 ri bad e^{s}/Me, In(3R)C, Sb e l(3)e
h136 ri e
h137 ri p^{p}
h138 ri p^{p} Ina (/ru h D InsCXF ca)
h139 ri p^{p} Inc l/ru h D InsCXF ca
h140 ri sbd e^{2}
h141 ro
h142 ro Bd ca/In(3R), l(3)a
h143 rs^{2}
h144 rsd
h145 ru
h146 ru h e^{s}
h147 ru h ri
h148 ru h ri p^{p} Inb
h149 ("threepl") ru h st p^{p} ss e^{s}
h150 ("rucuca") ru h th st cu sr e^{s} ca
h151 ("ruPrica") ru h th st cu sr e^{s} Pr ca/Me,T23
h152 ("rupes") ru h th st p^{p} cu sr e^{s}
h153 ru h th st p^{p} H e^{s} ro/ C(3)x, M(3)x e^{s} C(3)x = In(3L+R)P
h154 ru st C3G e^{s} (iso 3) (b sp)
h155 ru st C3G sr e^{s}
h156 ru tra p/ru h D InsCXF e
h157 ru^{g} jv se by
h158 ry
h159 ry^{2}
h160 Sb/In(3LR)Ubx^{101}
h161 Sb bx^{D}/Xa,T23
h162 Sb H/In(3R)C, cd
h163 Sb Ubx/Xa
h164 Sb^{Spi}/In(3LR)Cx
h165 sbd^{2} bx^{3}
h166 se
h167 se h
h168 se ss
h169 se ss k e^{s} ro
h170 se^{51j}
h171 se rt^{2} th/Me,InL
h172 ("separated arms of 3" Dubinin) T3L·41;4R·3R/1 InLP Dfd InRP l
h173 sep,InLR ri p^{p}
h174 sep,InLR ri p^{p} Sb/Me, InL Dfd InRC e l3e
h175 sep,InLR ri p^{p} Sb/Me, InL InRC e l3e
176 Ser/In(3R)C, e l(3)e
h177 snb
h178 sr
h179 sr gl
h180 ss
h181 ss bx
h182 ss bx Su^{2}-ss
h183 ss bxd k e^{s}/Xa
h184 ss ca (iso 1953)
h185 ss e^{s} (iso 1953)
h186 ss^{a}
h187 ss^{a}-40a
h188 ss^{a}-B
h189 ss^{A},In3/Sb bx^{D}
h190 st
h191 st c 3G ca/TM1, Me ri Sb^{1} (sp^{2})
h192 st in ri p^{p}
h193 st Ki p^{p}
h194 st Sb^{r} e^{s} rv ca
h195 st sr H^{2} ca/In(3R)P^{W}, st 1(3)W ca
h196 st^{sp}
h197 st^{54i} ri p^{p}
h198 su ve ru ve h th
h199 su-ve ru ve bv (h? th?)
h200 su-ve ru ve h th
h201 $(sp^{2};)$ su^{2}-Hw bx bxd/ Me,Ins ri Sb^{1}

h202 th
h203 th cu sr e^s ro ca
h204 th st cp
h205 th st pb p^p/In(3LR)Cx
h206 th st pb p^p kar su^2-Hw jvl ss bx sr gl/TM1, Me ri Sb^1
h207 tra/Me,T23
h208 Tri/ru h D InsCXF
h209 tt wo
h210 tx
h211 Ubx e^4/Payne, Dfd ca (Ubx=bx^D)
h212 ve
h213 ve bv (iso 1957)
h214 ve ca (iso 1953)
h215 ve h th
h216 ve R/In(3L)P, gm
h217 ve R D^3 bx^D (e^s?) Pr ca/InLP Dfd InRP ca
218 ve R D^3 Sb^{Spi} Bd^G/InsP
h219 ve st (iso 3)
h220 ve st sbd
h221 W
h222 W Sb/InsCXF.
h223 wk/Payne, Dfd ca
h224 wo
h225 Xa,T23 ca/e^s cd ro cmp ca

Chromosome 4

i1 ar/ey^D
i2 bt
i3 bt ey^R sv^n
i4 bt^D/ci^D
i5 Cat/ci^D
i6 Ce^2/spa^{Cat}
i7 ci^D/ey^D
i8 ci ey^R
i9 ci ey^R sv^n
i10 ci gvl bt
i11 ci gvl ey^R sv^n
i12 ci^{361}
i13 ci^W
i14 ey
i15 ey^2
i16 ey^4
i17 ey^D/ci^D
i18 gvl
i19 4-sim/ci^D
i20 gvl ey^R
i21 gvl ey^R sv^n
i22 gvl sv^n
i23 M(4)/ey^D
i24 pol
i25 spa
i26 sv^{35a}
i27 sv^{de}/ey^D
i28 sv^n
i29 Tp4:Y (Edmondson)/ 4-sim ♂ & 4-sim ♀

Multiple Chromosomes

X,2 (j1-j15)

j1 Bld,T12 InCyR/sc^2 pn; II^+
j2 ("scute twelvepl") y sc^5; al dp sc^{19i} b pr cn vg c a px bw mr sp/al^2 Cy pr Bl cn^2 L^4 sp^2
j3 sc^8 f In49 v; bw^{VA}/L^2 l (iso Y,X,2)
j4 X·Y InEN v y; S dp Sp cn/dp^{tx} Cy cn (no free Y)
j5 y; S Sp cn/dp^{tx} Cy cn
j6 y ac; sc^{19i}/S^2 Cy
j7 y f:=; bw^{VA}/L^2 l
j8 f^{56e} & y f:=; cn bw
j9 y f:=; Cy,Ins cn^2/Gla,InLR
j10 y f:=; dp^{tx} Sp cn bw/S^2 Cy cn bw
j11 y f:=; net bw sp
j12 y Hw In49 m g/y sc^{S1} B InS; net bw sp
j13 y sc^{S1} In49 v sc^8; dp^h b bw/dp^{txl} Cy pr Bl cn^2 L^4 sp^2
j14 y v f^x:f^{+ih}; bw^{VA}/L^2 l
j15 y^2 t^2; cn bw

X,3 (j16-j24)

j16 sc w B^{S3}.Y^S & y f:= (B^{S3} Del.-Inser, into 3)
j17 sn^3; Mw/l InLP InRP l
j18 ("Tam tester 1") y f:=; D^3 Sb/InLP Dfd InRP ca
j19 ("Tam tester 2") y sn oc ♂ & Y^+/y sc^{S1} B In49 l sn^{X2} sc^8/lJ1 sc^{J1} oc ptg ♀; ru h D InsCXF/Me,Ins ri Sb^1
j20 ("Tam X3") TX3,red ♂ & y f:= ♀
j21 ("Tam X3sn") sn TX3,red ♂ & y f:= ♀
j22 w^a ♂ & y v f·= ♀; tra/D InsCXF
j23 y Hw In49 m g/y sc^{S1} B InS; ru bw
j24 y f:=; su-ve ru ve bv (h? th?)

X,4 (j25-j27)

j25 y sc^{S1} InS sc^8 ♂ & y·=♀; ci ey^R
j26 y f:=; Cat/ci^D
j27 y f:=; spa

2,3 (j28-j104)

j28 al b cn sp/al^2 Cy Bl cn^2 L^4 sp^2; ru
j29 "apl"/Cy sp; ru h InsCXF ca/Sb InRMo
j30 b; p^p
j31 bw; e
j32 bw; ru h st D^3 ri InRC e l3e/Me,Ins ri Sb^1
j33 bw; ru h ri
j34 bw; ss
j35 bw sp; ru h D^1 ri InRC e l3e/Me,Ins ri Sb^1
j36 bw; st
j37 c; e
j38 cn bw; ri ej39 cn bw; ru h th ri e^s
j39 cn bw; ru h th ri e^s
j40 cn crs/al^2 Cy lt^3 pr Bl cn^2 L^4 sp^2; e^s
j41 cn crs/Cy pr Bl cn^2 L^4 sp^2; ve (iso)
j42 Cy/Pm; ru h D Ins CXF ca/InLP Dfd InRP ca
j43 Cy/Pm; st (iso X,2,3)
j44 De bw; ro
j45 dp cu bw/Cy Fl cu^2 L^4 sp^2; h ri e^s
j46 dp^{o3} cn bw; ru h D^3 ri In3RC e l3e/Me,Ins ri Sb^1
j47 dp^T Sp cn bw/S^2 Cy cn bw; ri e
j48 dp^T Sp cn/S^2 Cy cn; ri e
j49 dp^T Sp cn/S^2 Cy cn; ru h D^3 ri InC e l3e/Me Ins ri Sb^1
j50 dp^{tx} Sp cn/S^2 Cy cn; ru h D InsCXF/Me Ins ri Sb^1
j51 dp^{tx} Sp cn/S^2 Cy cn; ru h D InsCXF Sb/Me,InL InC e l3e
j52 dp^{tx} Sp cn/S^2 Cy cn; sep ri pP Sb/Me InL InC e l3e
j53 dp^{tx} Sp ms ta cn crs/S^2 Cy pr Bl cn^2 L^4 sp^2; e^s
j54 dp^{tx} Sp pr cn^2/S^2 Cy cn^2; Me·InL InC e l3e/ru h CXF Sb
j55 dp^V; vo^3
j56 dp^{V2} cn bw; h ri e^s (iso 7/57)
j57 fes ms cn sp/dp^{txI} Cyc05 pr cn^2; h ri e^s/Me Ins ri Sb^1 (iso 7/57)
j58 InNSL InNSR mr/al^2 Cy pr Bl lt^3 cn^2 L^4 sp^2; ri^{53j}
j59 ("iser 1") S Sp (crs)/Cy InL lt^3; Me Ins ri Sb^1/Bd^G
j60 ("iser 2a") ms cn rm sp/al^2 Cy lt^3 pr Bl cn^2 L^4 sp^2; ru h D InsCXF/ve th l
j61 ("iser 2b") dp b cn c P-/al^2 Cy lt^3 pr Bl cn^2 L^4 sp^2; ru h D Ins CXF/D; J e P^i
j62 M33a/al^2 Cy pr Bl cn^2 L^4 sp^2; ru
j63 ms sp/Cy pr Bl cn^2 L^4 sp^2; ri^M (iso)
j64 net bw mr crs; Dl H e P^i/ru h D InsCXF (low iso 7.57)
j65 net bw mr crs/dp^{txI} Cy O pr cn^2; ve bv/Me,Ins ri Sb^1
j66 net dp sp/dp^{txI} Cy,O pr cn^2; Me,Ins ri Sb^1/ve bv
j67 ("Pale e") dp b cn c P$^-$/Cy cn^2; e P^i/e P^i
j68 ("Pale H") dp b cn c P$^-$/Cy cn^2; p^{56} Dl H e P^i/p^{56} In3R l
j69 ("Pale Indp") IndpT23 b P$^-$·Dl H e P^i/dp b Pm^1; Sb In3R
j70 S fes Sp T23B D^3 ri Sb/Cy cn^2 sp^2; InsCXF
j71 S fes Sp T2301 ms cn mr crs D^3 st ri InC e l3e/al^2,InMis Cy pr Bl cn^2 L^4 sp^2; Me Ins ri Sb^1
j72 S fes Sp T2301 ms cn mr crs D^3 st ri InC e l3e/dp^{txI} Cy,Ins05 pr cn^2; Me,Ins ri Sb^1
j73 ("sifter O") S Sp P- T23,InsCXF/dp^{txI} Cy,Ins05 pr cn^2; Dl H e P^i
j74 S Sp cn/dp^{txI} Cy cn; h ri e^s
j75 S Sp cn/dp^{txI} Cy cn; Me,InL InRC e l3e/ru h D Sb InsCXF
j76 S Sp cn/dp^{txI} Cy cn; ru h D^3 ri InRC e l3e/Me,Ins ri Sb^1
j77 S Sp cn/dp^{txI} Cy cn; ru h e^{11}
j78 S Sp cn/dp^{txI} Cy,Ins05 pr cn^2; h ri D^3 InC e l3e/Me,Ins ri Sb^1
j79 S Sp cn/dp^{txI} Cy,Ins 05 pr cn^2; ru h D InsCXF/Me,Ins ri Sb^1
j80 S Sp cn bw/dp^{txI} Cy cn bw; h ri e^s
j81 S Sp cn bw/dp^{txI} Cy cn bw; ru h D^3 ri InRC e l3e/Me,Ins ri Sb^1
j82 S Sp ms ta cn crs/dp^{txI} Cy pr Bl cn^2 L^4 sp^2; e^2
j83 Sp T23M1/dp^{txI} InsCy,05 pr cn^2; Me,Ins ri Sb^1
j84 sp; ru h D InsCXF/Me,InL InRC e l3e
j85 S Sp T23B/dp^{txI} Cy,Ins05 pr cn^2; Me,Ins ri Sb^1
j86 T23B cn bw InC e l3e/al^2 In,Mis Cy cn^2 L^4 sp^2; Me,Ins ri Sb^1

j87 T23B cn bw InRC e l3e/Cy,Ins05; ru h D InsCXF (InAM?)
j88 T23B cn bw D^3 ri/dp^{txI} Cy,Ins05 pr cn^2; Me ri Ins Sb^1
j89 ta/Cy Bl cn^2 L^4 sp^2; ru ri (iso)
j90 ta sp/Cy Bl cn^2 L^4 sp^2; jv (iso)
j91 ta sp/Cy cn^2 L^4 sp^2; ru (iso)
j92 ("TIn") dp^{txI} Cy,Ins05 pr cn^2 T23 Me,Ins ri Sb^1/S Sp cn; ru h D^3 st InRC e l3e
j93 (2;4) "apl" l/Cy cn^2 sp^2; IV-sim/ci ey
j94 (2,4) bw; ci^D/IV-sim
j95 (3,4) bv; Cat/ci^D
j96 (X,Y,2) Y:bw^+/y v & sc ct^n oc ptg car·y ct^1,In In49 sn^{x2}; bw
j97 (X,Y,3) ("multi- ") X·Y InEN y; st (no free Y)
j98 (X,Y,3) X·Y y; st (no free Y, no In)
j99 (X,Y,3) sc^8·Y/X·Y InEN y; ru h D InsCXF/ru tra p
j100 (X,Y,3) X·Y InEN In49 y; st (no free Y)
j101 (X,Y,4) sc^8·Y/X^+ & y f:=; ci gvl ey^R sv^n
j102 (X,Y,4) Tp^4:Y/X·Y InEN In49 v y; ci gvl ey^R sv^n
j103 (X,Y,4) Y^S·InEN y·Y^L: 4 & y·= ; ci ey^R (no free Y)
j104 (X,Y,4) Y^S·InEN y·Y^L sc^8 y^+; ci ey^R (no free Y)

X2,3 (j105-j112)

j105 ("MI") y^{Si} sc^8 InS y^3P; al^2 Cy lt^3 cn^2 sp^2/dp b Pm^1; ru h D InsCXF ca/Sb In3R
j106 ("Pale") w^e; P^-/Cy; P^i/P^i
j107 y^{Si} sc^8 B f In49 v; T23B D^3 st ri InC e l3e/dp^{txI} Cy,Ins05 pr cn^2; Me,Ins ri Sb^1 (select AT)
j108 y f:=; cn bw; e
j109 y Inr9 v; bw; e
j110 y sc^{S1} f In49 v sc^8; bw; e
j111 y^{Si} sc^8 B f In49 v; bw; e
j112 y sc^{S1} In49 sc^8; bw; st pP (to cross by sc^8·Y/y B for losses, l's & T's)
j113 (X,2,4) ("scar") sc t^2 v f car; Cy/bw; ey
j114 (Y,2,3) sc^8·Y:bw^+; dp^{v2} cn bw; h ri e^s (iso 7/57) (Cy Bl cn^2 L^4 sp^2)
j115 (Y2,3) sc^8·Y:bw^+; fes ms cn sp/dp^{txI} Cy,Ins05 pr cn^2; h ri e^s/Me,Ins ri Sb^1 (iso 7/57)
j116 (Y2,3) sc^8·Y:bw^+; net bw mr crs/dp^{txI} Cy,Ins05 pr cn^2; Me,Ins ri Sb^1 (iso 7/57) (ve bv)
j117 (Y,2,3) sc^8·Y:bw^+; net dp sp/dp^{txI} Cy,Ins05 pr cn^2; ve bv/Me,Ins ri Sb^1
j118 (Y,2,3) Y:bw^+; Me,T23/dp^{txI} Cy cn^2 bw sp
j119 (2,3,4) Cy/bw; e; ci^D/IV-sim
j120 (2,3,4) bw; e; ci ey^R

X,Y,2,3 (j121-j131)

j121 ("Multipare") sc^8·Y/y sc In49 B^{M1}; twl bw; st^{54i}
j122 ("Multipare R") sc^8·Y/X^{c2} y & y f:= ; twl bw; st^{54i}
j123 ("Taxy") sc^8·Y/y sn oc & sc^8·Y/y In49 sn^{x2} B^{M1}/y oc lz·Y^S ; twl bw; st^{54i}
j124 ("y s cn bw e") Y^{Lc}/y s·Y^S/y sc^{S1} B f In49 v sc^8; cn bw; e
j125 ("y cn bw e") Y^{Lc}/y·Y^S; cn bw; e
j126 sc^{VI}·Y^S/y In49 v f·Y^L; bw; e
j127 X·Y InEN In49 y; cn bw; e (no free Y)
j128 X·Y InEN In49 y; cn bw; ro (no free Y)
j129 (X,2,3,4) y f:=; bw; e; ci ey^R
j130 (X,Y,2,3,4) Y^S·InEN In49 y·Y^L; cn bw; e; ci ey (no free Y)
j131 (X,Y,2,3,4) Y^S·InEN In49 y·Y^L ♂ & "snocty" ♀ ; cn bw; e; ci ey^R (no free Y)

non-lethal "tumorous" stocks

m1 bw tu
m2 tu^{50j}
m3 tu^{51m}
m4 tu^h
m5 vg tu

SALT LAKE CITY, UTAH: UNIVERSITY OF UTAH
Department of Genetics

Note: Stock list unchanged. See DIS 33, p.53

SYRACUSE, NEW YORK: SYRACUSE UNIVERSITY
Department of Zoology

Several wild strains, each derived from a single inseminated female.
Several polygenic crossveinless (cve) strains.

TUSCON, ARIZONA: UNIVERSITY OF ARIZONA
Department of Zoology

w	dp	bw; e; ey
y	vg	T(3,4)A96,ca^2
y; w; ec; f	ss	S/Cy; D/c3X
b		

ARGENTINA

Buenos Aires: Atomic Energy Commission, Section of Genetics

Note: This list was copied from DIS 31, pg. 51 and DIS 32, pg. 40.

Wild Stocks

W1 Leningrad
W2 Buenos Aires
W3 Oregon-R

Chromosome 1 (homozygous)

Xa1 In49 B^{M1}
Xa2 y fa wy^2 g^2
Xa3 y t^2 v f
Xa4 y v
Xa5 y w f
Xa6 y w sn^3 f
Xa7 y^2 v f car
Xa8 y^{3P}, In B
Xa9 sc^6 car
Xa10 sc^8 B
Xa11 sc^8 bb w^a
Xa12 y^{S1} sc^8
Xa13 sc^{V2}, Inh
Xa14 sc^{S1} B InS w^a sc^8
Xa15 sc^{S1} In49 sc^8
Xa16 y sc^{S1} In49 w sc^8
Xa17 X·Y In EN v y (no free Y)
Xa24 B^W
Xa25 g^W
Xa26 w^{mR7aH1}
Xa27 y w sn^3
Xa28 y sc^{S1} sc^8
Xa29 X·Y y In49 v f car (no free Y)
Xa30 sc^8·Y/y_{sc^4} w sc^8 (sc^8·Y in ♂ & ♀)
Xa31 Y^{Lc}/X·Y^S

Chromosome 1 (attached-X)

Xb1 lz & y f:=
Xb2 sc ct^6 car & y f:=
Xb3 sc t^2 v f & y f:=
Xb4 y ac sc v & y f:=
Xb5 y ac t^2·Dp(y^+ ac^+ sc^{S1}) & y f:=
Xb6 y ct^6 f & ac^3 w^a ct f·=
Xb7 y ct^6 t^2 v f car & y f:=
Xb8 y·Dp(y^+ sc^{V1}) & y f:=
Xb9 y In49 sn^{X2} bb^1 & y f:=
Xb10 y sc lz^g v f & y f:=
Xb11 y $w^{m258-18}$ T^2 v f & y f:=
Xb12 sc^8 B In49 & y f:=
Xb13 sc^8 B In49 m & y f:=
Xb14 sc^8 car f In49 v & y f:=
Xb15 sc^8 Tu w^a & y f:=
Xb16 y^{S1} sc^8 B f In49 v w^a & y f:=
Xb17 y^{S1} sc^8 f Ins w^a & y f:=
Xb18 sc^{S1} f In49 v w & y f:=
Xb19 y sc^{S1} B f In49 v & y f:=
Xb20 sc^{L8} sc^8 & y f:=
Xb21 sc^{S1} B In49 sc^8 & y f:=
Xb22 y sc^{S1} B In49 sc^8 & y f:=
Xb23 sc^{S1} At In49 v w^a sc^8 & y f:=
Xb24 sc^{S1} f In49 v w^a sc^8 & y f:=
Xb25 sc^{S1} f InS y^{3P} & y f:=
Xb26 Y^L/f·Y^S & sc v f·=
Xb27 y^3·Y^L/y·Y^S & y f:=
Xb28 Y^S/g^2 B·Y^L & y f:=
Xb29 Y^S·Y^S 2/y v f·Y^L & y f:=
Xb30 sc^{V1}·Y^S/y In49 v B·Y^L & y f:=
Xb36 sn^3 v B^{M1}, In & y w f:=

Xb37 spl rb cx & y f:=
Xb38 Tu & y f:=
Xb39 y ac sc pn & y f:=
Xb40 y ct^6 f & ac^3 w^a ct f·=
Xb41 y sc^4 B InS w^a sc^8 & y f:=
Xb42 ("snoc") X·Y InEN ptg oc sn^5 & sc ct^n oc ptg car y In49 sn^{x2})·♀ (no free Y)
Xb43 ("X·Y ") Y^S·X InEN y·Y^L sc^8 y^+ & y^2 su-w^a w^a bb·+ (no free Y)
Xb44 ("X·Y By") Y^S·X InEN B y·Y^L & y^2 su-w^a w^a bb·= (no free Y)
Xb45 sc^8·Y/y B ♂ & y f·=♀
Xb46 Y^{Lc}/w oc ptg·Y^S ♂ & Y^{Lc}/y^2 X^+·w^a InS B sc^8 ♀ (tandem X·X giving rings)
Xb47 oc ptg Tu & y f:-
Xb48 w^e sn & y f:=
Xb49 w^e sn B & y f:=
Xb50 sc^{S1} In49 w sc^8 & y f:=

Chromosome 1 (balanced)

Xc1 N^8/y Hw In49 m g
Xc2 oc ptg^3·Dp(y^+ sc^{S1})/ClB
Xc3 oc ptg Tu/sc^{S1} fu In49 sc^8
Xc4 ras^4 m/ClB
Xc5 sc ct^n oc car/y In49 sn^{x2}·B^S
Xc6 sd f^{ex}/y sc^8 B f In49 v
Xc7 y sc^{S1} B In49 ct-l lz^s/w sn^{5s} bb
Xc8 ClB/w^e sn
Xc9 y sl^2 bb^-, In/InAM
Xc10 ct^6 v dy g f/InA99 sn^{33f}
Xc11 f fu/ClB
Xc12 un Bx/InAM ptg^4

Chromosome 1 (multiple loci)

Xd1 y ac sc pn w rb cm ct^6 ras^2 v g^2 f car & y f:=; sc^{19i}/Cy, InL lt
Xd2 y pn w cm ct^6 sn^3 oc ras^2 v dv g^2 f od car sw/y sc^{S1} B In49 v
Xd3 sc^{S1} car f In49 v/y ac sc pn w rb cm ct^6 sn^3 ras^2 v dy g^2 f car
Xd4 Y^{Lc}/y sn^5 oc v·Y^S ♂ & Y^{Lc}/sc^{J1} pn w rb cm ct^6 oc ras^2 v dy g^2 f od car sw/y sc^{S1} B In49 sn^{x2} sc^8 ♀
Xd5 y^3·Y^L/y^2 oc lz·Y^S ♂ & y^3·YL/y ac sc pn w rb cm ct^6 sn^3 oc ras v m g^2 f car/sc^{S1} B In49 lz^s sc^8 ♀
Xd6 lJ1·Y/lJ1 sc^{J1} In49 B^{M1}/y ac pn w ec rb cm ct^6 sn^3 oc ras^2 v dy g^2 f Tu car
Xd7 $lJ1^+$·Y/lJ1 $sc^{J1(+)}$In49 v f^{S1} B^{M1}/y sc^{S1} car odsy f g^2 dy v ras^2 sc^3 ct^6 cm rb ec w pn l sc^8

Chromosome 2

II-1 ab^2 ms ta crs/Cy pr Bl cn^2 L^4 sp^2
II-2 al b cn sp (iso, 1954)
II-3 cn bw sp
II-4 net b cn crs/dp^{txI} Cy pr Bl lt^3 cn^2 L^4 sp^2 (iso, 1955)
II-5 al dp b pr cn vg c a px bw sp/S^2 Cy lt^3 pr Bl cn^2 L^4 sp^2
II-6 Y:bw^+/X^+; cn bw
II-7 Y^c:bw^+/X^+; bw
II-8 c (iso 2,3)
II-10 ast ho
II-11 b pr
II-12 bw^D

Chromosome 3

III-1 e
III-2 ru h th st p^P cu sr e^s

Chromosome 4

IV-1 Cat/gyl ey^R
IV-2 ci ey^R
IV-3 spa
IV-4 sv^n

Multichromosomal

M1 w^a ♂ & y v f·= ♀; tra/D Ins CXF
M2 y sc^4 InS w^a; S sc^{19i} Bl/Cy L^4 sp
M3 sc^{19}-/lJ1 sc^{J1}; fes sc^{19i} b pr/Cy dp^{txI} pr cn^2
M4 Y^{Lc}/X·Y^S & y v f·=; e
M5 Y^{Lc}/X·Y^S; bw; e
M6 y sc^5; al dp sc^{19i} b pr cn vg c a px bw mr sp/al^2 Cy pr Bl cn^2 L^4 sp^2
M7 y^2 t^2; cn bw
M8 bw; e
M9 y^{S1} sc^8 InS y3P; al^2 Cy lt^3 cn^2 sp^2/ dp b Pm^1; ru h D InsCXF ca/Sb In3R
M10 w^e; P-/Cy; P^i/P^i
M11 y f:=; cn bw; e
M12 sc t^2 v f car; Cy/bw; ey
M13 Y bb^-/v; bw^{VA}/Bl L^2
M14 ("tester 1") y ac pn w rb wy^2 g^2 & y f:=; sc^{19i}/Cy
M15 ("tester 2") y^2 w^a cm wy^2 g^2 car & y f:=; sc^{19i}/Cy
M16 ("tester 3") y rb cm ras^2 g^2 & y f:=; sc^{19i}/Cy
M17 y ac sc pn w spl rb cx. & y f:=; (sc^{19i}(b pr)/)
M18 sc^8 B InS w^a sc^4 & y f:=; sc^{19i}/Cy cn^2
M19 y sc^- (rein. sc^{8-4}) w^a InS bb & y f:=; sc^{19i}/Cy lt cn^2
M20 Y^c:bw^+/X^{c2} y f; bw (ring OK '57)
M21 sc^8·Y:bw^+/ac^3; b bw
M22 Y^{Lc}/X·Y^S; bw

AUSTRALIA

Brisbane, Queensland: University of Queensland

Note: This list was copied from DIS 33, pg. 55

Wild Stocks

1 Oregon R-C

Chromosome 1

2 sc cv v
3 w
4 y/B

Multichromosomal

5 e^{11} dp
6 y; Cy/Pm, ds^{33k}, H/Sb

AUSTRIA

Vienna: Institut für allgemeine Biologie

Note: This list was copied from DIS 28, pg 51

Wild Stocks

1 Oregon-S
2 Oregon-R-c (Df(2)Ore)
3 Oregon-R-c P
4 8 strains from different places in Austria

Chromosome 1

12 Ax^{Oslo}
13 B
14 BB
15 B w^{bf}
16 ClB/dl-49 m^2 g^4
17 car
18 cv
19 fa^n
20 lz w^{bf}
21 pn^2
22 spl^2
23 v
24 w
25 w^{bf}
26 w^{bl}
27 w^{ch} wy
28 w^e
29 w^h
30 y
31 y w
32 y ec ct^6 v wy^2 car
33 y v f
34 y/f
35 bo

Chromosome 2

36 b pr vg a sp
37 bs (4 different alleles)
41 bw
42 cn
43 al dp
44 ex
45 j
46 L^2/Cy
47 vg

Chromosome 3

48 ca
49 c3G
50 e
51 e cu
52 gl
53 jv se
54 ru se h st bv
55 ru h th st cu sr e^s ca
56 Sb H/Payne
57 se e^{11}
58 Ser/In(3R)C, e l(3)e
59 ss^{a-F}
60 st

Chromosome 4

61 ey^2

Multichromosomal

62 Bx; J
63 v; cn bw
64 y v f/+; bw; e wo ro; ey^2

Aberrations

65 Df(1)N^8/y Hw dl-49 m^2 g^4
66 Df(2)vg^S, cn/Cy, al^2 lt^3 L^4 sp^2
67 Dp(1;f)135 y^2; In(1)sc^8, Df(0+ac) wa sc^8
68 In(2LR)Gla/Cy
69 T(2,3)bw/Cy

BRAZIL

Curitiba, Paraná: Universidade do Paraná, Faculdade de Dilosofia, Ciências e Letras, Laboratório de Genética

Note: This list was copied from DIS 32, pg 43.

Wild Stocks

Boa Esperança, Minas Gerais
Buenos Aires, Argentina
Campina Grande, Paraíba
Cosmópolis, São Paulo
Cuiabá, Mato Grosso
Curitiba, Paraná
Florianópolis, Santa Catarina
Gaspar, Santa Catarina
Goiânia, Goiás
Gruta, Argentina
Irati, Santa Catarina
Lins, São Paulo
Paranaguá, Paraná
Petrolina, Pernambuco
Ponta Grossa, Paraná
Salvador, Bahia
Santa Felicidade, Paraná
São Paulo, São Paulo
Teixeira Soares, Paraná
Uberlândia, Minas Gerais
Xapecó, Santa Catarina

CANADA

Toronto: University of Toronto

Note: Inbred stocks of Marvin Barr Seiger (see DIS 32, pg. 156)

Oregon-R: 291 generations of inbreeding as of 60k28
Ives Oregon-R: 311 generations
M Oregon R: 284 generations
P_1I Oregon R: 332 generations
2b Oregon R-C: Lost generation 256
f Oregon R: 221 generations
y Oregon R: 220 generations
Canton-S: 97 generations

Ives Oregon R: mass culture, extracted from inbred stock at generation 300
f Oregon-R: mass culture, extracted from inbred stock at generation 200
y Oregon-R: mass culture, extracted from inbred stock at generation 200

Multichromosomal

1 yf:=; bw; e; pol (1;2;3;4)
2 yv/FM6; Deb^D; $\frac{HnHu}{TM3}$ (1;2;3)
3 yv/v; bw^D; $\frac{Hu}{Ubx}$ (1;2;3)
4 $\frac{yv}{yv}$; $\frac{sp\ bw^D}{SM5}$; $\frac{Sb}{TM3}$; (1;2;3)
5 bw; st (2;3)

FRANCE

Lyon: Faculté des Sciences, Laboratoire de Zoologie

Note: This stock list copied from DIS 29, pg. 51.

Wild Stocks Champetières (inbred) Lyon

GERMANY

Göttingen: Max-Planck-Institut für Tierzucht und Tierernährung

Note: This stock list copied from DIS 33, pg.60

Wild Stocks

1 normal (Berlin wild)

Chromosome 1

2 br ec rb
3 ClB/+
4 sc^8 Y/y fx sc^8 y/X^{c2} y v
5 svr
6 w^e
7 w^{ch} wy
8 w^{co} v f
9 w^{co} y f
10 w sn^3 B
11 y w bb

Chromosome 2

12 fes lt L^4/Cy al^2 lt^3
13 fj px sp
14 lgl cn bw/Cy cn bw L^4 sp^2

Chromosome 3

15 pb/C Mé SB C
16 ss^a

Chromosome 4

17 ci^D/+
18 ey^D/+

Multichromosomal

19 w; j; e^{11}; ey^2
20 Cy/Pm ds^{33k}; H/C Sb
21 L Cy/+; C Mé Sb C/+

Hamburg: Zoologisches Staatsinstitut und Zoologisches Museum

Note: This stock list copied from DIS 32:49.

Wild Stocks

1 Oregon-S

Chromosome 1

2 w^1
3 y/f

Chromosome 2

4 bw
5 dp b
6 Pm L Cy

Chromosome 3

7 cu
8 e cu
9 Sb/H Payne

Chromosome 4

10 ey^2

Multichromosomal

11 bw; cu
12 bw; ey^2
13 bw; cu; ey^2
14 cu; ey^2
15 y; ey^2

Triploid

16 y^2 sc w^a ec/FM4, y^{31d} sc^8 dm B
17 y^2 sc w^a ec/FM4, y^{31d} sc^8 dm B; bw; cu; ey^2

Hamburg-Eppendorf: Universitäts-Frauenklinik, Strahlenbiologische Abteilung

Note: This stock list copied from DIS 28:56

Wild Stocks

1 normal (Berlin wild)

Chromosome 1 (X)

2 ClB/+
3 sc^{S1} B InS w^a sc^8
4 sc^8 Y/y f x sc^8 Y/X^{c2} y v
5 w
6 X^c/ClB

Multichromosomal

7 cn; ss

Attached-X

8 y

GREAT BRITAIN

Bayfordbury, Hertford, Herts, England: John Innes Horticultural Institution

Note: This stock list copied from DIS 33:62.

Wild Stocks

1 Bayfordbury
2 Hampton Hill
3 Samarkand
4 Teddington

Inbred Lines

5 Bayfordbury (A)
6 Bayfordbury (B)
7 Oregon
8 Samarkand

Chromosome 1

9 v
10 w
11 y w

Chromosome 2

12 b pr vg

Multichromosomal

13 Cy L^4/Pm; H/Sb

Glasgow, Scotland: University of Glasgow, Department of Genetics

Note: This stock list copied from DIS 32:51.

Wild Stocks

1 Florida-4 (inbred)
2 Oregon-K (inbred)

Chromosome 1

3 B
4 gt w^e
5 sc w ec ev
6 sc w^{bl} ec cv
7 sn^3
8 v
9 w
10 w^{FF33}
11 w^a
12 w^{bf2}
13 w^{co}
14 w^e
15 w^h
16 w^{sat}
17 w sn^3
18 w sn^3 B
19 w^t
20 y w^e ec
21 w^{1P} z(zeste)
22 w^{14G2} z(zeste)

Attached-X

23 y v f

Chromosome 2

24 b cn vg
25 bw

Chromosome 3

26 E

Multichromosomal

27 y v f/v; bw^{VA}/Bl L^2
28 bw; e
29 bw; v
30 sn^3; cu sr e^s p^p

Inversions

31 Muller-5

Harwell, Berks., England: Medical Research Council, Radiobiological Research Unit

Note: This stock list copied from DIS 33:63.

Wild Stocks

1 Oregon-K

Inbred lines

2 Crianlarich (186 gens.)
3 Kaduna (90)
4 lt (18)
5 Nettlebed (268)
6 Oregon-R (336)
7 Oregon-S (229)
8 stw (18)
9 Wild Edinburgh (260)

Chromosome 1

10 Muller-5

Chromosome 2

11 a px
12 al dp b pr c px sp/Cy
13 b
14 b el
15 bw
16 bw pd
17 cn
18 Cy/Bl L^2
19 ds dp
20 el
21 hk pr
22 ho ed cl
23 lt
24 ltd
25 ltd cn
26 net
27 pd
28 pr
29 stw
30 stw lt

INDIA

Calcutta: Indian Statistical Institute

Note: This list copied from DIS 34:37-38.

Wild Stock

1 Canton-S

Chromosome 1

2B
3 Bx^3
4 cv
5 Df(1)N^8/dl-49 y Hw m^2 g^4
6 ec
7 f
8 In(1)Cl $sc^?$ $t^?$ v^+ $sl^?$
B^{36d}/dl-49 y Hw v m^2 f
9 m
10 v
11 w
12 w^a
13 w^{bf}
14 w^{bl}
15 w^{co}
16 w^e
17 w^h
18 w^i
19 y
20 y^2

Chromosome 2

21 bw
22 cn
23 dp
24 ho
25 px
26 vg
27 vg bw

Chromosome 3

28 cu
29 e^s
30 Ly/D^3
31 ry
32 se
33 ss^a

Chromosome 4

34 ci^W
35 ey^2

Multichromosomal

36 Cy/Pm D/Sb

Calcutta: University of Calcutta, Department of Zoology

Note: This stock list was copied from DIS 32:53.

Wild Stocks

a1 Canton-S
a2 Oregon

Chromosome 1

b1 y Hw 49 v^{o} m^{2}, f/ClB^{36d}
b3 x^{c-2} y/y w f
b6 y

Chromosome 2

c2 L^{4}
c3 vg
c4 ho

Chromosome 3

d1 se h
d2 ss^{a}
d3 Ly/D^{3}
d4 e^{s}

Chromosome 4

e1 ey^{2}
e2 ci^{W}

Multichromosomal

f1 Cy/Pm; D/Sb

Hiroshima: Hiroshima University, Zoological Laboratory

Note: This stock list was copied from DIS 33:68.

Wild Stocks

1 Kandy, Ceylon
2 Hiroshima
3 Matsuyama
4 Miyakonojio
5 Naze
6 Oregon-R
7 Samarkand
8 Sapporo
9 Taichu, Formosa

Chromosome 1

10 BB
11 w

Chromosome 2

12 S/Cy E-S
13 vg

Chromosome 3

14 e

Multichromosomal

15 Muller-5; Cy/Pm; Sb, e/Ubx^{130} e
16 y; bw; e; ci, ey^{R}

Mitaka, Tokyo: International Christian University

Note: This stock list was copied from DIS 33:70.

Wild Stocks

Oregon-S
Tokyo

Mutants

Muller-5
w
w^{a}
w^{e}
y
y w m f
cu
e

NETHERLANDS

Leiden: Rijksuniversiteit, Laboratorium voor Stralengenetica

Note: This stock list was copied from DIS 33:74-75.

Wild Stocks

1 Oregon-K

Chromosome 1

2 car^{26-48} f^{3N} 7 y f:=
3 cm ct^{6} sp^{3} & y w f:=
4 cv cm^{28-4} f^{3N} & y f:=
5 cn f^{3N} & y f:=
6 f B odsy car
7 fu^{ff}/ClB
8 g^{2}
9 pn
10 pn^{27-9} cv v f^{3N}
11 ras dy
12 rb
13 rb^{27-4} v f^{3N} & y f:=
14 sc^{S1} B In-S w^{a} sc^{8} (Muller-5)
15 sw

16 "tester 1-y" y ac pn w rb wy^2 g^2 & y f:=
17 "tester 2-y" y^2 w^a cm wy^2 g^2 car & y f:=
18 "tester 3-y" y rb cm ras^2 g^2 & y f:=
19 un^4 Bx^2 & y f:=
20 w sn B
21 y ac sc pn w rb cm ct^6 ras^2 v g^2 f car & y f:=; sc^{19i}/Cy, In-L lt ("Maple")
22 y sc^{S1} B In-49 sn^{x2} sc^8/oc ptg
23 y sc^{S1} In49 v sc^8
24 y w In-49 f
25 y w^a cv v f
26 X^{c2} y B & y f:=

Chromosome 2

37 b pr vg
38 dp
39 dp b cn bw
40 dp stw^3 bw
41 dp^{Th} Cy, In-L pr cn^2 In-Cy R-O/Ins-NSL Ins-NSR px sp ("Cy Oster")
42 dp^{Th} Cy cn bw/S Sp cn bw
43 fes ms cn sp/net dp^{txI} Cy b pr Bl lt^3 cn^2 L^4 sp^2
44 ls dp^T Sp ms ta cn crs/S^2Cy lt^3 pr^+ Bl cn^2 L^4 sp^2
45 net bw mr crs/al^2, In-Mis dp^{txI} Cy Bl cn^2 L^4 sp^2
46 S fes Sp ms cn mr crs/al^2 In, Mis dp^{txI} Cy pr Bl cn^2 L^4 sp^2

Chromosome 4

47 Ci^D/spa^{Cat}

Altered Y's

27 X·Y In-EN v ptg oc sn^5 & y sc t^2 v f car·= (no free Y)
28 X·Y In-EN y; st (no free Y) ("Multi♀")
29 l $J1^+$·Y/l J1 $sc^{J1(+)}$ In-49 ptg oc B^{M1}/ y^{S1} sc^{S1}In car odsy f g^2 dy v ras^2 sn^3 ct^6 cm rb ec w l pn sc^8 ("Maxy-new")
30 l $J1^+$·Y/y sc^4 B v^{41b} & y sc^4 B v^{41b}/y w In-49 lz^S
31 l $J1^+$·Y/y In-49 f^{S1} B^{M1}
32 sc^8·Y/y sc^{S1} B f In-49 v & y f:=/ sc^8·Y
33 sc^8·Y/y Hw In-49 v ptg oc f^{S1} B^{M1}/ y^{Si} sc^{S1}, In car odsy f g^2 dy v ras^2 sn^3 ct^6 cm rb ec w l pn sc^8
34 sc^8·Y/y In-49 B; bw^D ♂ & y f:=; bw^D ("Multi♂")
35 Y^{Lc}/X·Y^S ("Sterilizer +")
36 Y^{Lc}/X·Y^S; cn bw; e ("Sterilizer cn bw e")

Multichromosomal

48 y sc^{S1} In-49 sc^8; dp b cn bw
49 cn bw; e
50 Cy/Pm; Cx, D/In(3R)Sb
51 y sc^{S1} In-49 sc^8; bw; st pP

Leiden: Rijksuniversiteit, Genetisch Laboratorium

Note: This stock list was copied from DIS 32:62
Only some unusual stocks are listed.

Chromosome 2

cn bw Dr/Pm
cn px bw Kr/Cy
crc cn/Pm

Chromosome 3

gs
h gs th

Triploid

♀ 3N:W/FM_4 & Y/FM_4 ♂

Utrecht: Rijksuniversiteit, Genetisch Instituut

Note: This stock list was copied from DIS 33:75-76.

Wild Stocks

1 Oregon-K

Chromosome 1

2 cm ct^6 sn^3 & y w f:=
3 cv car^{26-48} f^{3N} & y f:=
4 cv f^{3N} & y f:=
5 fu/ClB
6 g^2
7 pn
8 ras dy
9 rb
10 rb^{27-4} cv v f^{3N} & y f:=
11 sc cv v f
12 sc^{S1} B In-S w^a sc^8
13 ("tester 1") y ac pn w rb wy^2 g^2 & y f:=; sc^{19i}/Cy

14 ("tester 2") y^2 w^a cm wy^2 g^2 car & y f:=; sc^{19i}/Cy
15 ("tester 3") y rb cm ras^2 g^2 & y f:=; sc^{19i}/Cy
16 w sn B
17 y sc^{S1} B In-49 sn^{x2} sc^8/oc ptg
18 y^{S1} sc^8 B f In-49 v
19 y w m B
20 y w^a cv v f

Altered Y's

21 X·Y·In-EN y; st (no free Y) ("Multi ♀").
22 $lJ1^+$·Y/lJ1 $sc^{J1(+)}$ In-49 ptg oc B^{M1}/y^{S1} sc^{S1}, In car odsy f g^2 dy v ras^2 sn^3 ct^6 cm rb ec w l pn sc^8 ("Maxy-new")
23 sc^8·Y/y Hw In-49 v ptg oc f^{S1} B^{M1}/y^{Si} sc^{S1}, In car odsy f g^2 dy v ras^2 sn^3 ct^6 cm rb ec w l pn sc^8
24 sc^8·Y/y In(1)49 B; bw^D ♂ & y f:=; bw^D ("Multi ♂")
25 Y^{Lc}/X·Y^S ("Sterilizer +")
26 Y^{Lc}/X·Y^S; bw ("Sterilizer bw")
27 Y^{Lc}/X·Y^S; dp ("Sterilizer dp")
28 Y^{Lc}/X·Y^S; cn bw; e ("Sterilizer cn bw; e")

Chromosome 2

29 b pr vg
30 bw
31 Bl L/Cy
32 dp
33 dp b cn bw
34 dp^{Th} Cy, In-L pr cn^2 In(2)Cy R-)/ Ins-NSL Ins-NSR px sp
35 dp^{Th} Cy cn bw/S Sp cn bw
36 J/In(2L0t, l(2)B

Chromosome 3

37 e
38 h ri
39 l tr/e In(3R) In(3L)
40 Mio/In(3R)Sb
41 St

Chromosome 4

43 ci
44 ci^D/spa^{Cat}

Multichromosomal

45 y sc^{S1} In-49 sc^8; dp b cn bw
46 w^a ♂ & y v f:=; tra/D Ins-CXF
47 cn bw; e
48 Cy/Pm; Cx, D/In(3R)Sb

Stocks selected for abnormal abdomen

49 (AA) DcxF/Mé Sb
50 (AA) Cx, D/In(3R)Sb

Deficiencies

53 Df(1)N^8/dl-49, y Hw m^2 g^4
54 Df(1)$N^{264-105}$ (dm)/dl-49, y Hw m^2 g^4
55 Df(1)N^{264-39} w^{ch}/FM4, y^{31d} sc^8 dm B

SOUTH AFRICA

Johannesburg: University of the Witwatersrand, Department of Zoology

Note: This stock list copied from DIS 33:78

Wild Stocks

1 Bethulie
2 Bloemfontein
3 Canton-S
4 Cape Town
5 Cedara
6 Florida
7 Graaff-Reinet
8 Inhaca Island
9 Johannesburg
10 Limpopo
11 Nelspruit
12 Oregon-R
13 Stanford Lake
14 Stellenbosch
15 West Rand
16 Zoutpansberg

Chromosome 1

17 bi ct^6 g^2
18 bo
19 B
20 car
21 car^2
22 cm
23 cm car
24 cm g^3 car
25 ct v
26 ct v dy g
27 ct^6
28 ec
29 ec ct^6 v g^3
30 f_5 B
31 f^5 m
32 f^5 v
33 g
34 g^2
35 g^3
36 m
37 pn^2
38 ras dy
39 ras^2
40 ras^3 m
41 rb
42 rb car
43 rb cm g^3
44 rb cm car
45 rb cx
46 rb g^3
47 rb g^3 car
48 sc ec cv ct^6 v g^2 f
49 sc ec cv ct^6 v g^2 f/FM3 y^{31d} sc^8 dm B l
50 svr w^a
51 v
52 v^{36f}
53 v g^3

54 w
55 w m
56 w m f
57 w^a
58 w^{a3}
59 w^{a4}
60 w^a rb
61 w^{bl}
62 w^{ch}
63 w^{co} sn^2
64 w^{col}
65 w^e
66 w^{e2}
67 w^{e3}
68 w^e car
69 w^e cm
70 w^e g^3
71 w^e r^b
72 w^e rb car
73 w^{sat}
74 w^t fw
75 w^w f^5
76 w^w rb 77 y
77 y
78 y g^4
79 y m
80 y pn
81 y rb
82 y w
83 y w m
84 y^2 su-w^a w^a bb
85 y^2 w^a w

Chromosome 2

86 albasp/Cy L^4 sp^2
87 al dp b pr Bl c px sp/ SM1 al^2 Cy sp^2
88 a sp^2
89 b
90 b pr
91 b pr cn
92 b pr cn a
93 bw
94 bw^{2b}
95 bw^4
96 bw^D
97 c px
98 cn
99 cn^{35k}
100 cn vg
101 cl
102 cl^{50a}
103 dke c
104 dp
105 lt std/cy sp^2
106 lt stw^3
107 ltd
108 pd
109 pr
110 pr^{42d}
111 sf^2
112 Su-H/Cy, pr
113 tk sf^2 abb
114 vg
115 vg^{dn}

Chromosome 3

116 ca
117 cd
118 cu Kar
119 D/Gl
120 e
121 e^s
122 e^s cd ro cmp ca/Xa, ca
123 ma fl
124 mah
125 p
126 p^p cu
127 p^p cu sr e^s
128 res
129 ru
130 ry
131 se
132 sr
133 st^{sp}
134 su^B-pr/In(3R)C, e; pr
135 th st
136 th st p^p

Chromosome 4

137 ci ey

Multichromosomal

138 bw; e
139 bw; e; ci ey
140 bw; ci ey
141 bw; st
142 Cy/Pm, ds^{33k}; H/ In(3R)Mo; sr
143 g^3; bw
144 g^3; st
145 g^3; st p^p
146 ras^2; st
147 rb; bw
148 rb; ry
149 rb; se
150 rb; st
151 car; ry
152 car; se
153 vg; se
154 w^e rb; se
155 w^w; cd
156 y; bw; e; ci ey

Attached-X

157 f B/su^{S2}-v-pr v
158 y/+
159 y^2 su-w^a w^a bb/y sc^{4L} sc^{8R}

Inversions

160 In(1)A99b
161 In(1)dl-49, y fa^n
162 In(1)rst^3, y rst^3 car bb
163 In(1)rst^3, y rst^3 g^3 car
164 In(1)w^{m4}
165 In(1)w^{m4}; bw
166 In(1)w^{m4}; st
167 Ins(1)sc^{S1}, S, sc^{S1} w^a B sc^8

Translocations

168 T(1;3)04, D/ClB
169 T(1;4)w^{m5}

SPAIN

Madrid: Centro de Investigaciones Biológicas, Laboratorio de Genética

Note: This stock list was copied from DIS 33:79

Wild Stocks

Madrid	Ribadeo	Ronda 10
Mallorca	Rocafort	Ronda 30

SWEDEN

Stockholm: University of Stockholm, Institute of Genetics

(From DIS 33:79-81)

Wild Stocks

1 Algeria
2 Canton-S
3 Djursholm 55
4 Florida
5 Karsnäs
6 Oregon
7 Stäket
8 Tunnelgatan
9 Örebro
10 Skaftö

Chromosome 1

11 B
12 bb
13 BB; sc^8 Y ♂ y f:=; sc^8 Y ♀
14 B car; sc^8 Y ♂ y f:=; sc^8 Y ♀
15 BB car; sc^8 Y ♂ y f:= sc^8 Y ♀
16 Bx^2
17 car
18 cm ct^6 sn^3 ♂ y f:= ♀
19 cv
20 cv sn
21 cv v B ♂ y f:= ♀
22 ct^6
23 ec ct v f
24 f
25 f B
26 f BB; sc^8 Y ♂ y f:=; sc^8 Y ♀
27 fa
28 fu; sc^8 Y ♂ y f:= sc^8 Y ♀
29 g^2 B
30 g f car ♂ y f:= ♀
31 lw^{29a} H_2/y Hw g In-49 m
32 lw^{47b} H_1/y sc^8 f In-49 V w^a
33 lz ♂ y f:= ♀
34 m
35 m f
36 Df(1)N^8/w^a
37 od car
38 pn
39 rb
40 sc
41 sc^8
42 sc cv
43 sc cv v f
44 sc cv v car
45 sc ec cv ct^6 v g f/FM3, y^{31d} sc^8 dm B l
46 sc t^2 v f Tu car ♂ y f:= ♀
47 sc^{S1} B In-S w^a sc^8
48 sc^{S1} B In-S w^a sc^8; y sc^8 Y
49 sn^3
50 v
51 w
52 w cv
53 w cv sn^3
54 w sn^3
55 w^a
56 w^{bf2}
57 w^{bl}
58 w^{ch2}
59 w^{co}
60 w^{col}
61 w^{ec2}
62 w^h
63 w^r sc^8 In-S
64 w^t
65 X^{c2} (closed-X)
66 y
67 y^2 eq; Df(Y)Y-bb
68 y^{329}
69 y^{3P}
70 y^4
71 y ac sc pn sn; sc^8 Y
72 y ac sc pn w rb cm ct^6 sn^3 ras^4 v m g f car/sc^{S1} B In-S w^a sc^8
73 y B^{267-47}
74 y ec ct^6 v f
75 y f car
76 y f Eb/sc^{S1} B In-S w^a sc^8 ♀ sc^{S1} B In-S w^a sc^8 ♂
77 y Hw m g f Eb/sc^{S1} B In-S w^a sc^8 ♀ sc^{S1} B In-S w^a sc^8 ♂
78 y Hw m f g car; sc^8 Y ♂ y f:=; sc^8 Y ♀
79 y rb ct^6
80 y sc
81 y sc w^e ec rb
82 y sc^8 B f In-49 v ♂ y f:= ♀
83 y sc^{S1} f In-49 m sc^8 ♂ y f:= ♀
84 y sc^{S1} In-49 v sc^8 ♂ y f:= ♀
85 y sc^{S1} In-S w^a sc^8
86 y v f·= ♀ y w ♂
87 y v f car·= ♀ sc v f car ♂
88 y v g f
89 y w sn^3
90 y w sn; sc^8Y
91 y w f Bx^2
92 y w spl sn
93 y w^a sn

Chromosome 2

94 a px sp
95 a px or
96 al b c sp
97 al sp b pr Bl c px sp/SM1, al^2 Cy sp^2
98 al dp b pr c px sp/al^2 Cy lt^3 L^4 sp^2
99 al dp b pr cn vg c a px bw mr sp/ S^2 Cy lt^3 pr^+ Bl cn^2 L^4 sp^2
100 al^2 Cy lt^3 L^4 sp^2/Pm
101 al S ast ho/Cy, E-S
102 al sp
103 b
104 b cn vg
105 b pr vg

106 Bl/In(2LR)dp
107 bw
108 cn bw
109 cn vg bw
110 Cy/Pm
111 dp b
112 dp b pr c px sp
113 ed Su^2-dx
114 ft
115 L^2/Cy
116 L^4
117 pr
118 px bw mr sp/ds^{33k} Pm
119 S^2 Cy pr Bl cn^2 L^4 bw sp/In-NSL In *NSR px sp
120 sca
121 sp
122 stw^3
123 vg

Chromosome 3

125 ca
126 D^3/In-P
127 e^s
128 gl
129 Hn^{r2}
130 ri^2
131 ro
132 ru Hn^{r2}
133 ru h st p^p ss e^s
134 ru se h st p^p ss e^s
135 ru h th st cu sr e^s Pr ca/Mé, T(2;3)
136 se ss k e^s ro
137 ss
138 st
139 st ss e^{11}
140 ve h th
141 ss e^{11}

Chromosome 4

142 ey
143 ci ey

Multichromosomal

144 cn bw; e^{11}
145 bw; st
146 rb cm ras^2 g^2; sc^{19i}/Cy ♂ f:= sc^{19i}/Cy ♀
147 sc cv v; ri
148 sp; th
149 T(1;2)B^{bd}/Cy ♀ M(2)e/Cy ♂
150 T(1;2)Bld/Cy
151 TX(16A1)4 B^s y w f:=
152 T(2;3)Met/Cy
153 lt/T(Y;2)A
154 y v; bw^{VA}/L^2 l
155 y; pr; ss
156 +•=; sv^n ♀ +; sv^n ♂

Uppsala: Botanik-Genetiska Institutionen, Lantbrukshogskolan

Note: This stock list was copied from DIS 32:65-66.

Wild Stocks

1 Algeria
2 Amherst-3
3 Bayforbury
4 Boa Esperanca, Minas Gerais, Brazil
5 Canton-S
6 Crimea
7 Curitiba, Paraná, Brazil
8 Florida
9 Gruta, Argentina
10 Hikone-R (resistant to BHC, DDT, parathione, nicotine)
11 Karsnäs
12 Kochi-R (resistant to parathione)
13 Oregon
14 Salvador, Bahia, Brazil
15 Samarkand
16 San Miguel, Buenos Aires, Argentina
17 Stäket
18 Tunnelgatan
19 Örebro
20 Örebro-R (resistant to parathione)

Chromosome 1

21 B
22 B/Y
23 BB car; sc^8 Y ♂ y f:=; sc^8 Y ♀
24 ct
25 cv
26 cv sn^3
27 Dp(1)w^a ♂ y w f:= ♀
28 ec
29 ec ct v f
30 f
31 f B od sv car
32 f BB; sc^8 Y ♂ y f:=; sc^8 Y ♀
33 f od car
34 In(1)w^{m4}
35 is
36 lz/ClB
37 ma-l y f:=
38 m f
39 sc^{S1} B InS w^a sc^8
40 sc^{S1} InS w^a sc^8
41 sc z w^{17G2} ec ct
42 sn^3
43 sp-w
44 su-w^a w^a

45 w
46 w ct
47 w cv
48 w cv sn^3
49 w sn^3
50 w^a
51 w^{aE}
52 w^a su-f ♂ y f:= ♀
53 w^{bf2}
54 w^{bf} f^5
55 w^{bl}
56 w^{ch} wy
57 w^{co}
58 w^{co} sn^2
59 w^e
60 w^{e2}
61 w^{e2} en-w^e ♂ y f:=♀
62 w^h
63 w^h ct
64 w^i yb
65 w^{sat}
66 y
67 y ac sc pn w rb cm ct^6 sn^3 ras^4 v m g f car/sc^{S1} B InS w^a sc^8
68 y ec ct v f
69 y f Eb/sc^{S1} B InS w^a sc^8
70 y^2 sc w^a w^{ch} fa ♂ y f:= ♀
71 y^2 sc w^i
72 y^2 sc w^i w^{ch} ♂ y f:= ♀
73 y^2 su-w^a w^a w^e ♂ y f:= ♀
74 y^2 w^a
75 y^2 w^a w
76 y z
77 z ec
78 z w^e ec
79 z w^{11} E4

Chromosome 2

80 bw
81 cn vg bw
82 Cy/Pm 83
83 Cy/S
84 fes lt^3/cy al^2 lt^3 L^4 sp^2
85 net
86 pr
87 S/NS, px sp
88 vg

Chromosome 3

89 D^3/InP
90 ri^2
91 ro
92 ru h st pP ss e^s
93 se
94 ss
95 st ss e^{11}

Chromosome 4

96 ey
97 sv^n

Multichromosomal

98 cr-u/Cy; (w^e)
99 Cy/S; D/InP
100 In(1)w^{m4} y^{511}; E-Var 4/Cy
101 In(1)w^{m4}; E-Var 5/Cy
102 In(1)w^{m4}; E-Var 8/Cy
103 L^2/+, sp; th
104 sp; th
105 T(2;3)bw^{VDe4}/Cy
106 T(1;4)w^{m5}
107 w^{ch}; Su-w^{ch}/Cy
108 y^{S1} sc^8 InS y^{3P}; al^2 Cy lt^3 sp^2/dp b Pm^1; ru h D^3 InCXF ca/Sb In(3R)

NEW MUTANTS

Report of A. B. Burdick

dy^{58k}: dusky-58k M.E.Krawinkel, 1958k. Spontaneous in an isogenic pol stock, W-160 pol. A bona fide new mutant since dy, the only other dy mutant known at the time dy^{58k} was discovered, was not present in stocks at this institution at that time, and the pol marker of the stock in which it occurred was present in the single mutant male observed. Recombination and complementation tests show that dy^{58k} is the right-most known element of the m-dy complex. It recombines with Df(1)259-4, with all m's except m^{D}, and not with dy. It appears to be the right-most element because it consistently shows higher recombination and more complementation with m-type mutants than does either m^{D} or dy. Wing length is shorter than dy, about the same length as the longer m-type mutants. Fertile in both sexes. RK1.

m^{59a}: miniature-59a M.E.Krawinkel, 1959a. Probably spontaneous in an isogenic wild-type background (W-126) which several generations before had been treated with about 50r of X-ray. Shows low complementation with m-mutants and high with dy-mutants. Recombines with Df(1)259-4 on its left and m on its right. No m-mutant (except Df(1)259-4) has been shown to be left of m59a. Female fertility very low; male fertile. RK2.

dy^{60k}: dusky-60k A.B.Burdick, 1960k. Spontaneous in a SM5, al^{2} lt^{V} Cy sp^{2}/da stock; isolated in uniform stock background so that we know that it could not have arisen as a contamination. Allele tested with m^{60l} and dy^{61a} (see DIS-35, new mutant report of P. T. Ives); shows high complementation with m^{60l} and low with dy^{61a}. Similar to other dy's; fully fertile in both sexes. RK1.

rk^{4}: rickets-4 R.C.Jackson, 1954. Originally called cq (creeper) in DIS-28, 1954. H. U. Meyer in DIS-32 reports cq to be an allele of Edmondson's rk. Therefore, cq is now rk^{4}.

r^{58a}: rudimentary-58a M.Burdick, 1958. Induced in mature wild-type sperm by 4000r X-ray. Resembles r (Morgan, 10f) and r^{9} (Bridges, 20b3), wings truncated, blistered, wing-veins, particularly L4, frequently interupted, marginal bristles sparse, somewhat longer, and disarrayed. Expression variable but does not overlap wild-type. Female entirely sterile. Recombination tested in 2894 flies to car and 974 to f giving confidence intervals that include 1-54.5, Morgan's locus for r. Functionally allelic with r^{9}. RK2.

Report of W. W. Doane

(This report supersedes that in DIS-34 inadvertently attributed to S. Counce.)
fs(2)adp: female sterile(2)adipose Counce, 1956. 2-83±. Pub. Doane, 1959, Genetics 44:506; developmental and physiological studies, Doane, 1960, Ph.D. Thesis, Yale Univ. Spontaneous in Kaduna wild stock maintained in Edinburgh. Adult fat body hypertrophies as cells become distorted by enormous oil globules. Abnormal fat bodies visible through body wall of 6-day old and older adults when submerged in 95% alcohol, then water. Adult corpus allatum of mated females hypertrophies. Females completely sterile, sterility autonomous. Eggs laid by homozygotes show meiotic and/or mitotic abnormalities and never develop beyond early cleavage stages. Males 78% fertile. Heterozygotes fertile, but females become sterile with age. Viability generally good, but longevity reduced; homozygotes with selective advantage under starvation; heterozygotes superior under desiccation. Average water content of well fed adults reduced, while percentage of lipids, as a function of dry body weight, is almost double that of wild type. Iodine numbers show greater degree of saturation of mutant lipid extracts than of wild type. RK2.

The mutant flag (fg), reported as a new mutant in DIS-34 and located approximately at 2-20±, has been more accurately located by linking it with a dumpy marker. The new cross-over data indicate it to be at 2-22±, and so it has been checked for allelism with the mutant spade (spd), located at 2-22.3±. The latter was obtained from the spd gt-4/SM5, al^{2} Cy lt^{V} sp^{2} stock at Pasadena, California. Spd is given

the rank of RK_5 in Bridges and Brehme because of its poor penetrance; fg is fully penetrant with an RK_1. None of the spd/sdp flies examined from stock bottles has shown wing effects comparable to those described for fg, and most of them resemble wild type. However, when spd gt-4/Cy females were crossed to fg/fg males (or the reciprocal cross made), all of the non-Cy offspring showed a wing effect which ranged from a slight shortening to a shape mid-way between the phenotypes of the two different mutants. It appears, therefore, that fg and spd are allelic and that the former should henceforth be referred to as spd^{fg} ($spade^{flag}$).

Report of J. L. Hubby

Recovery of another rosy allele. Ins(3RC;3LP) Sb e^s/ry^2 was shown to have a rosy phenotype and greatly reduced xanthine dehydrogenase activity (Hubby and Forrest, 1960). A double crossover between st and Sb was recovered from this inversion which proved to have a rosy phenotype when homozygous or when in combination with rosy or $rosy^2$. No crossovers have been recovered among approximately 5000 progeny from females carrying $rosy^2$ and this mutant. This mutant has therefore been designated $rosy^3$.

Homozygous ry^3 males show traces of isoxanthopterin and uric acid in their testes, hence ry^3 is a "leaky" mutant with respect to the products of xanthine dehydrogenase. Thus far no satisfactory procedure has revealed convincing evidence of xanthine dehydrogenase activity in extracts of this mutant.

Report of P. T. Ives.

dy^{61a}: $dusky^{61a}$. Ives, 61a24. Like dy. Induced by 1 kr γ radiation in an Oregon-R/rucuca sperm which was deposited on day 5 of an exhaustive mating schedule. Functional allelism to dy established by A. B. Burdick who found normal recombination (with g) and good fertility and fecundity in both sexes. RK1

m^{60l}: $miniature^{60l}$. Ives, 60l26. Like m. Induced by 1 kr γ radiation in an Oregon-R/rucuca sperm which was deposited on day 6 of an exhaustive mating schedule. Functional allelism to m established by A. B. Burdick who found recombination with g somewhat reduced and fertility and fecundity good in both sexes. RK1

sd^{58d}: $scalloped^{58d}$. Ives, 58d14. Strongest sd allele, with vg-like wings and weak vg-like effects on halteres and bristles. Induced by 1 kr γ radiation in an Oregon-R sperm which was deposited on day 7 of an exhaustive mating schedule. Functional allelism to sd indicated by strap shaped wing in sd/sd^{58d}. Recombination normal in y ct^6 ras^2 regions but reduced by 80% between ras^2 and f, suggesting a small In associated with sd^{58d}. Not studied cytologically. Genetic tests show no T. Relative frequency of sd^{58d} is sometimes low when competing with non-sd flies but a pure line breeds well. Combines readily with vg alleles and vg deficiencies. RK-2.

Report of James F. Kidwell

Dfd^{r60J} - Spontaneous recurrence in sixth generation of full sib mating derived from Princeton wild stock. Expression varies from both eyes absent to wild type. Expression may be asymmetrical. Penetrance varies from 75 to 100 per cent. Penetrance increased by selection for reduced eye. About 5 per cent of flies $Dfd^{r60J}/+$; ey/+ exhibit deformed phenotype.

Report of H. W. Lewis

$alpha^{-1}$. The report of this mutant was erroneously included in DIS-34 under the report of E. B. Lewis.

Report of A. Schalet

$ma\text{-}l^2$: $maroon\text{-}like^2$ Schalet, 1961. From X-rayed y ct^6f. $Dp(y^+sc^{v1})$ ♂♂ mated to $ma\text{-}l^1$ females. Hemizygous male and homozygous female are viable and fertile. Brownish-red eye color is like $ma\text{-}l^1$ and $ma\text{-}l^{bz}$. $ma\text{-}l^2$ does not complement $ma\text{-}l^1$

$ma\text{-}l^1$, $ma\text{-}l^{bz}$ or $ma\text{-}l^3$ (see below).

ma-l^3: maroon-like^3 Schalet, 1961. From X-rayed y ct^6f. Dp(y^+sc^{vi}) ♂♂ mated to $ma\text{-}l^1$ females. Hemizygous males inviable. In a small-scale test the absence of crossovers between v and $ma\text{-}l^3$ indicate possible association with a gross rearrangement. Heterozygotes of $ma\text{-}l^3$ with $ma\text{-}l^1$ or $ma\text{-}l^2$ are mutant in appearance and similar in color to the homozygotes of the latter alleles. A chromosome carrying v and $ma\text{-}l^3$ heterozygous with y v f $ma\text{-}l^{bz}$ chromosome also shows a mutant appearance with respect to maroon-like.

Report of A. H. Sturtevant

Correction:

spa^{pol}: sparkling-poliert. The mutant poliert of Hadorn (Rickenbacher, DIS 27: 59) is an allele of spa, giving when crossed to spa, heterozygotes with eyes slightly more extreme than spa. spa^{pol} is probably the most useful recessive in the fourth chromosome.

Report of V. Tinderholt

Cyg: The Curly gene Tinderholt, 58f 2-6.1±.9. The Curly gene is generally inseparable from the inversion, In (2L) Cy, but has been crossed out by increasing the frequency of double crossovers. This was accomplished by using females carrying the complex inversions FM6; SM5 (which includes In (2L) Cy); TM3 Ser, as the three major chromosomes. The dominant character associated with this gene when free of the inversion is identical to the one resulting from its presence within the inversion. The symbol Cyg was chosen because Cy generally is used to designate the gene-inversion combination.

The Curly gene was first localized between heldout (ho, 2-4.0) and echinoid (ed-11.0). Out of 2,489 flies, there were 1,127 Cyg ed and 1,241 wild type non-crossovers. The crossovers consisted of 64 Cyg and 57 ed giving a value of 4.9±.9 crossover units to the left of ed. RK1.

TM3 Sb Ser: Third Multiple 3 with Stubble and Serrate See DIS 34:51, Report of E. B. Lewis, and DIS 34:53-54, Report of V. Tinderholt. The third chromosomal multiple rearrangement, TM3 Sb Ser carries the genes bx^{34c} Sb ri p^p sep and e^s. The Stubble gene apparently arose from a new mutation and not from a rare double crossover as was suggested in the previous DIS note. The gene bx^{34c}, 0.6 crossover units from Sb still remains in the chromosome. A double crossover would have removed bx^{34c} as Stubble was inserted.

LINKAGE DATA

Report of D. J. Nash and E. C. Keller, Jr.

The dominant sex-linked miniature wing mutant reported in DIS-34 appears to be identical to m^D. The mutant is allelic to both m and dy, and in its interactions with m and dy cannot be distinguished phenotypically from the interactions involving m^D. No crossovers have been found among over 5,000 progeny which might have shown a crossover between this allele and m^D.

STOCK LISTS

PITTSBURGH, PENNSYLVANIA: UNIVERSITY OF PITTSBURGH

(From DIS 33:106)

D. persimilis

Wild Stocks

Yosemite National Park, el. 8000' (White Wolf)
Whitney (4 strains)
Klamath (4)
Mendocino (4)

Yosemite, el. 10,000' (Timberline)j
Whitney (4)
Klamath (4)
Mendocino (4)

Mather, California: 15 strains incl. Wh, Kl, and St

Chromosome 2

Dl cd
cd

Multichromosomal

Dl or Cy

ROCHESTER, NEW YORK: THE UNIVERSITY OF ROCHESTER

(From DIS 33:106)

D. busckii

There are more than 300 stocks of D. busckii in the collection of J. Krivshenko. They include X-chromosomal and autosomal mutants (with visible effects), in various combinations, as well as a number of special stocks in which lethals are associated with chromosomal aberrations. Dominant and recessive markers, associated with inversions or other types of chromosomal aberrations, are available for each chromosome. There are also 17 strains from geographically remote populations.

D. persimilis

Wild Stocks: 12 strains from various localities in Western North America.

Chromosome 1

se

Chromosome 2

ss^a

Chromosome 3

Delta or Cy

D. pseudoobscura

Chromosome 1

ct se ll sp tt

Chromosome 2

up bx Ba gl (in)/lethal

Chromosome 3

or

Chromosome 4

in hk j

Multichromosomal

Ba Cy sparky

Wild strains homozygous for gene arrangements on the third chromosomes, as follows:

Standard
Mather (6 strains)
Pinion (6)

Arrowhead
Bryce (8)
Ferron (13)
Gunnison (16)
Leman Cave (13)
Mono Lake (10)
Pinion (6)

Chiricahua
Mather (6)
Pinion (6)

114 inbred lines of the above Arrowhead stocks in F_{19}-F_{28} of sib mating
33 strains from various geographical localities in Western North America
12 strains each from: Helena, Calif. Lone Pine, Calif.
Sebastopol, Calif. Wild Rose, Calif.
Hopeland, Calif. Placerville, Calif.

Other Species

D. fuliginea: Rochester, N.Y. (1 strain)
D. funebris: Rochester, N.Y. (1)
D. hydei: 1 strain heterozygous for an inversion on the second chromosome including sections 2-D1 through 2-G2 (collected at Raleigh, N.C., August 1954). 1 wild strain from Rochester, N.Y.
D. immigrans: Rochester, N.Y. (1)
D. miranda: Big Basin (1)
D. repleta: Rochester, N.Y. (2)
D. robusta: Rochester, N.Y. (1)
Megacelia scalaris: Princeton, N.J. (1)

FRANCE

Gif sur Yvette (S et O): Centre National de la Recherche Scientifique, Laboratoire de Génétique Evolutive (From DIS 27:70)

D. funebris

Wild type from Challuz
Wild type from Chatenay-Malabry
Wild type from St Mandé

D. simulans

Wild type from Dr. Haldane
Wild type from Dr. Sturtevant
Wild type from South Africa

Mutant types: Net Pm
se (?)

ITALY

Pavia: University, Institute of Genetics
(Type Culture Collection of Drosophila Species)
(From DIS 33:113)

D. acanthoptera (1 strain)
D. affinis (1)
D. algonquin (1)
D. ambigua (2)
D. athabasca (1)
D. azteca (1)
D. bifasciata
Wild Stocks (3)
Mutants: a
f
g
ob
or
y
sex-ratio (2)
D. busckii (1)
D. buzzatii (1)
D. cameraria (1)
D. cardini (1)
D. dscabibi Burla (1)
D. duncani (1)
D. funebris
Wild Stocks (3)
Mutants: Bb^y
b w s; st
cn
co np; st
co no; st, cu
co np/$Stub^y$
cu; st
ev
miniature-vermilion
np
Pch
sn^2; st
sn^2 w y np
st 45h
Va
w
w N np
w sn np
D. gibberosa (1)
D. guttifera (1)
D. helvetica (1)
D. hydei (1)
D. immigrans (1)
D. kuntzei (1)
D. latifasciaeformis (3)
D. lativittata (1)
D. lebanonensis (1)
D. littoralis (1)
D. miranda (1)
D. montium (1)
D. narragansett (1)
D. nitens
Wild Stocks (3)
Mutants: or
y
D. obscura (2)
D. persimilis (1)

D. Phalerata (1)
D. prosaltans (1)
D. pseudoobscura (2)
D. putrida (1)
D. repleta (1)
D. robusta (1)
D. setifemur (1)
D. simulans (2)
D. spinofemora (1)
D. subbadia (1)
D. subobscura
Wild Stocks (1)
Homozygous standard:
Esperöd
Kussnächt
D. transversa (1)
D. tripunctata (1)
D. tristis (2)
D. victoria (1)
D. virilis (1)
D. willistoni (1)
D. yakuba Burla (1)
Zaprionus vittiger (1)
Z. tuberculatus (1)

KOREA

Kwangju: National Chunnam University, Laboratory of Genetics

(From DIS 31:104)

D. virilis

Wild Stocks	Inversion
Japan (2 strains)	In(X)Spd
Korea (5 strains)	

Other Species

D. alboralis (1 strain)
D. angularis (1)
D. arizonensis (1)
D. auraria (5)
D. bifasciata (2)
D. bizonata (3)
D. busckii (3)
D. buzzatii (1)
D. cheda (3)
D. coracina (3)
D. duncani (1)
D. hamatofila (1)
D. hayashii (2)
D. histrio (2)
D. immigrans (3)
D. lacertosa (2)
D. lutea (3)
D. mirim (1)
D. mulleri (1)
D. nigromaculata (3)
D. repleta (1)
D. suzukii (2)
D. testacea (1)
D. unispina

SPAIN

Barcelona: Centro de Genética Animal y Humana del Consejo Superior de Investigaciones Cientificas

(From DIS 33:117)

D. ambigua: several Spanish stocks
D. bifasciata: Pavia (Italy)
D. busckii: Barcelona
D. cameraria: Cantonigrós (Spain)
D. funebris: several Spanish stocks
D. immigrans: Barcelona
D. kuntzei: Cantonigrós (Spain)
D. mercatorum mercatorum: Barcelona
D. mercatorum pararepleta: Jijuca (Brazil)
D. phalerata: several Spanish stocks
D. repleta: New Haven (Conn.); Berlin
D. simulans: Barcelona
D. subobscura: several Spanish stocks; mutant stocks
D. transversa: Montnegre (Spain)
Parascaptomyza disticha: Barcelona

Irwin H. Herskowitz, Editor

D. = Drosophila; D.m. = Drosophila melanogaster

Abd-el-Wahab, A. 1959. The determination of facet number in D.m. II. Effect of acid amides. J. Genet., 56:437-442.
1959. The determination of facet number in D.m. III. Effect of ureids and acid amides on roughoid. J. Genet., 56: 475-481.
1959. The determination of facet number in D.m. IV. Effect of amino acids on roughoid. J. Genet., 56: 482-485.

Abd-el-Wahab, A., and Sirlin, J.L. 1959. Nuclear RNA and hormone production in the ring gland of D. Exp. Cell Res., 18:301-312.

Alderson, T. 1960. Mechanism of formaldehyde-induced mutagenesis. The uniqueness of adenylic acid in the mediation of mutagenic activity of formaldehyde. Nature, Lond., 187:485-489.
1960. Significance of ribonucleic acid in the mechanism of formaldehyde-induced mutagenesis. Nature, Lond., 185:904-907.

Alexander, M.L. 1960. Dose rate effects and genetic recovery with cobalt-60 γ-rays in atmospheres of exygen and nitrogen. (Abstr.) Rec. Genet. Soc. Amer., 29:53; and Genetics, 45:971.
1960. Radiosensitivity at specific autosomal loci in mature sperm and spermatogonial cells of D.m. Genetics, 45:1019-1022.

Alexander, M.L., Bergendahl, J., and Brittain, M. 1959. Biological damage in mature and immature germ cells of D. virilis with ionizing radiations. Genetics, 44: 979-999.

Altenburg, E., and Browning, L.S. 1960. Photoreactivation of mutations induced in D. by different doses of ultraviolet light. (Abstr.) Rec. Genet. Soc. Amer., 29:54; and Genetics, 45:972.
1960. The relative proportion of whole-body mutations versus fractionals in D. (Abstr.) Rec. Genet. Soc. Amer., 29:54-55; and Genetics, 45:972-973.
1961. The relatively high frequency of whole-body mutations compared with fractionals induced by X-rays in D. sperm. Genetics, 46:203-211.

Anders, G. 1960. Papierchromatographischer Nachweis von höheren, nichtflüchtigen Fettsäuren bei D.m. Rev. Suisse Zool., 67:171-183.

Aradi, M.P. 1959. Die Drosophiliden-Fauna des Karpatenbeckens. Rov. Közlem., 12: 409-426.

Arnold, L.L. 1957. Breeding D. in disposable paper containers. Amer. Biol. Teach., 19:248-251.

Auerbach, C. 1960. Chemical mutagens in animals. Abh. Deutsche Akad. Wiss., Berlin, Jahrgg., 1960:1-13.
1960. Discussion on the mutagenic effects of alkylating agents. Genet. Res., Cambr., 1:332.

Auerbach, C., and Sonbati, E.M. 1960. Sensitivity of the D. testis to the mutagenic action of mustard gas. Z. Vererbungsl., 91:237-252.

Auerbach, C., and Westergaard, M. 1960. A discussion of mutagenic specificity. Abh. Deutsch. Akad. Wiss. Berlin Klasse Medizin, pp.116-123.

Auerbach, C., and Woolf, B. 1960. Alpha and beta loci in D. Genetics, 45:1691-1703.

Avio, C.M., and Tarozzi, G. 1959. Further studies in the oviposition of D. grown in two different electric conditions. Riv. di Biol., 51:145-152.

Baglioni, C. 1959. Genetic control of tryptophan peroxidase-oxidase in D.m. Nature, Lond., 184:1084-1085.
1960. Genetic control of tryptophan pyrrolase in D.m. and D. virilis. Heredity, 15:87-96.

Baker, W.K. 1960. Genetic control over the somatic differentiation of eye pigments in D. (Abstr.) Anat. Rec. 138:332.

Bakker, K. 1959. Feeding period, growth, and pupation in larvae of D.m. Ent. Exp. et Appl., 2:171-186.
1960. An analysis of the competition for food between larvae of D.m. Ent. Ber., 20:244-255. (In Dutch with English summary.)

Barigozzi, C. 1960. New data on unexpected genetic heterogeneity obtained from a tumourous stock in D.m. Atti A.G.I., 5:71-78.
1960. Trasformazioni inaspettate nel genotipo di ceppi con tumore di D. Acc.

Naz. Lincei, Quaderno 47, 'Evoluzione e Genetica', 183-203.
Barigozzi, C., Castiglioni, M.C., and Di Pasquale, A. 1960. A complex genotype controlling the production of melanotic tumours (pseudotumours) in D. Heredity, 14: 151-162.
Bateman, A.J. 1959. Polygenic mutations affecting viability. (Abstr.) Heredity, 13: 414-415.
1960. Relative biological efficiency of 20 MV and 4 MV radiations. V. Induction of dominant lethal mutations in D. sperm. Brit. J. Radiol., 33:278-280.
Baumiller, R.C., and Herskowitz, I.H. 1960. The relation between phenotypic detriment of heterozygous mutations and genetic load in D. (Abstr.) Rec. Genet. Soc. Amer., 29:56-57; and Genetics, 45:974-975.
1961. The effects of X ray induced, euploid and near-euploid mutants in heterozygous condition upon developmental stages of D.m. Ph.D. Thesis, Saint Louis Univ. St. Louis, Mo., 72 pp.
Beardmore, J.A. 1960. Developmental stability in constant and fluctuating temperatures. Heredity, 14:411-422.
Beardmore, J.A., Dobzhansky, Th., and Pavlovsky, O. 1960. An attempt to measure the fitness of monomorphic and polymorphic populations of D. pseudoobscura. Heredity, 14:19-33.
Becker, H.J. 1960. Variegation in the zeste eye color alleles and its bearing on gene action during the development of the eye of D.m. Genetics, 45:519-534.
Belgovskii, M.L., Abeleva, E.A., and Potekhina, N.A. 1959. The frequency of induced dominant lethals in different stages of spermatogenesis in D. Akad. Nauk SSSR. Dok., 128:1279-1282. (In Russian.)
Belitz, H.J. 1959. Einige Ergebnisse und Probleme der Chemogenetik. Naturwiss. Rund., 12:458-461.
Bell, A.E. 1957. Genetic and phenotypic changes in D. populations under selection. Proc. Sixth Poultry Breeders Roundtable, pp. 81-103.
Bell, A.E., and Moore, C.H. 1958. Further comparisons of reciprocal recurrent selection with conventional methods of selection for the improvement of quantitative characteristics. (Abstr.) Proc. X International Cong. of Genetics, 2:20-21.
Bender, H.A. 1960. Studies on the expression of various singed alleles in D.m. Genetics, 45:867-883.
1960. Studies on the expression of various singed alleles in D.m. Diss. Abstr., 20:3476.
Bender, H.A., and Green, M.M. 1960. Effects of a suppressor of the lz^{34k} allele in D.m. (Abstr.) Anat. Rec. 138:333-334.
1960. Phenogenetics of the lozenge loci in D.m. I. Effects of a suppressor of lz^{34k}. Genetics, 45:1563-1566.
Bennett, J. 1960. A comparison of selective methods and a test of the pre-adaptation hypothesis. Heredity, 15:65:77.
1960. A simplified method of D. culture for the classroom. Educational Bull. Service, Northern Illinois Univ., DeKalb, Ill.
Bennett, J., Capek, R., Kallstedt, T., and Moisand, R. 1960. Venation polymorphism and genetic variability in D.m. Meigen. Science, 132:1399-1440.
Bereskin, B., and Hollander, W. F. 1960. Bithorax and heterosis effects on egg yield in D.m. Amer. Nat., 94:405-411.
Bergerard, J. 1958. La polyploidie somatique chez les animaux. Ann. Biol., 34:119-132.
Bernstein, N., and Goldschmidt. E. 1961. Chromosome breakage in structural heterozygotes. Amer. Nat., 95:53-56.
Bert, G.R. 1960. Fixed heterozygosity and fitness in D.m. populations under strong natural selection. (Abstr.) Rec. Genet. Soc. Amer., 29:57; and Genetics, 45:975.
Bochnig, V. 1960. Über Beziehungen von modifikatorisch und genetisch bedingten Eigenschaftsänderungen zur Widerstandsfähigkeit gegenüber DDT bei D.m. Z. angew. Zool., 47:323-334.
Bochnig, V., Lüers, H., and Winterfeld, G. 1960. Die mittlere Lebensdauer von D.m. nach einmaliger Röntgenbestrahlung mit hohen Dosen. Zool. Beiträge, N.F., 5:367-372.
Bodmer, W.F., and Parsons, P.A. 1960. The initial progress of new genes with various genetic systems. Heredity, 15:283-299.
Bonnier, G. 1959. Note on the relative sensitivity to irradiation damages in homozygous D.m. with regard to their capacity of producing offspring. Hereditas, 45: 675-676.

1961. Experiments on hybrid superiority in D.m. I. Egg laying capacity and larval survival. Genetics, 46:9-24.
1961. Experiments on hybrid superiority in D.m. II. Rate of development from egg hatching to eclosion. Genetics, 46:85-91.
Bösiger, E. 1960. Sur le rôle de la sélection sexuelle dans l'évolution. Experientia, 16:270-273.
Braver, G. 1960. The influence of an adjacent inversion breakpoint on unequal crossingover in the Bar region of D.m. (Abstr.) Rec. Genet. Soc. Amer., 29: 59; and Genetics, 45:977.
Breese, E.L., and Mather, K. 1960. The organisation of polygenic activity within a chromosome in D.m. II. Viability. Heredity, 14:375-399.
Brehme-Warren, K. 1959. The second edition of "The mutants of D.m." by C.B. Bridges and K. Brehme-Warren. Biol. La. (Cold Spr. Harb.), 69:51-52.
Brenner-Holzach, O., and Leuthardt, F. 1959. Untersuchungen zur Biosynthese der Pterine bie D.m. Helvet. Chim. Acta, 42:2254-2257.
Brncic, D. 1958. Evolución en el Grupo mesophragmatica del género D. Biologica, 26:3-46.
1961. Integration of the genotype in geographic populations of D. pavani. Evolution, 15:92-97.
Brosseau, G.E.(Jr.) 1960. Genetic analysis of the male fertility factors on the Y chromosome of D.m. Genetics, 45:257-274.
1960. V-type position effects influencing the action of the Bar locus in D. (Abstr.) Rec. Genet. Soc. Amer., 29:61; and Genetics, 45:979.
Brosseau, G.E., Nicoletti, B., Grell, E.H., and Lindsley, D.L. 1961. Production of altered Y chromosomes bearing specific sections of the X chromosome in D. Genetics, 46:339-346.
Brown, E.H., and King, R.C. 1961. Studies on the expression of the transformer gene of D.m. Genetics, 46:143-156.
Brown, Wm.P., and Bell, A.E. 1960. Alternative methods of selection applied to plateaued populations of D.m. (Abstr.) Rec. Genet. Soc. Amer., 29:61; and Genetics, 45:979.
Browning, L.S., and Altenburg, E. 1960. The relatively high rate of unequal crossing over between Brownex and white in D. (Abstr.) Rec. Genet. Soc. Amer., 29:61-62; and Genetics, 45:979-980.
Burdette, W.J. 1960. Alteration of mutation frequency by treatment with actinomycin D. Science, 133:40.
Burdick, A.B. 1959. Wild-type iso-allele hypothesis of mutation in polygenic systems. Proc. Genet. Soc. Japan, 31:293-294.
Burton, L. 1960. Carcinogenic activity of larval donor extracts in D. Diss. Abstr. 20:3029.
Capenos, M.R. 1959. The origin of lethality in certain second chromosome recombinations of D. persimilis. Diss. Abstr., 20:1543-1544.
Capps, A.S. 1961. The effects of nitric oxide on radiation damage in D. virilis and D.m. Genetics, 46:123-127.
Carfagna, M., Solima, A., Cioffi, E., and Virzo, A. 1960. Osservazioni sul comportamento di alcuni dominanti letali in popolazioni artificiali di D.m. II. Variazioni dell'eterosi a seconda della costituzione genetica. Rend. Acc. Naz. Lincei, 28:1-9.
Carlson, E.A. 1960. Is complementation mapping of complex loci valid for D.? (Abstr.) Rec. Genet. Soc. Amer., 29:62; and Genetics, 45:980.
Carpenter, J.M. 1960. Studies on the role of wild yeasts in fluctuating populations of D. (Abstr.) A.S.B. Bull., 7:25.
Carson, H.L. 1959. Genetic conditions which promote or retard the formation of species. Cold Spr. Harb. Symp. Quant. Biol., 24:87-105.
1960. Survival of newly-induced chromosome aberrations in experimental populations of D.m. (Abstr.) Rec. Genet. Soc. Amer., 29:62-63; and Genetics, 45:980-981.
Caspari, E.W. 1960. Richard Benedict Goldschmidt. Genetics, 45:1-5.
Cavalcanti, A.G.L., Falcão, D.N., and Castro, L.E. 1958. The interaction of nuclear and cytoplasmic factors in the inheritance of the "sex-ratio" character in D. prosaltans. Publ. Faculdade Nac. Filos., Univ. Brasil, Sér. Cient., No. 1, 54 pp. (In Portugese and English.)
Chandley, A.C., and Bateman, A.J. 1960. Mutagenic sensitivity of sperm, spermatids, spermatocytes and spermatogonia in D.m. Heredity, 15:363-376.

Chen, P.S., and Schläpfer, Th. 1959. Zur Atmungsphysiologie der Mutante letal-meander (lme) von D.m. Arch. Jul. Klaus-Stiftg., 34:240-245.
Chovnick, A., Weisbrot, D., and McMullen, J. 1959. Study of a complex locus in D. Biol. Lab. (Cold Spr. Harb.), 69:15-24.
Clark, A.M. 1960. The mutagenic activity of some pyrrolizidine alkaloids in D. Z. Vererbungsl., 91:74-80.
Clarke, J.M., Smith, J. Maynard, and Sondhi, K.C. 1961. Asymmetrical response to selection for rate of development in D. subobscura. Genet. Res., Cambr., 2:70-81.
Clayton, F.E. 1959. The effect of lozenge psuedoalleles on eye pigmentation in D.m. III. Pigment development during pupal differentiation. Genetics, 44:1041-1052.
1960. Determination of D. karyotypes from adult males. Evolution, 14:134-135.
Colwell, R.R., and Burdick, A.B. 1959. Uptake and effect on crossing-over of ethylenediamine-tetraacetic acid (EDTA) in D.m. Nucleus, 2:125-130.
Coomes, R.K., and Bennett, J. 1959. Use of the World Health Organization mosquito test kits with DDT resistant D. Trans. Ill. St. Acad. Sci., 52:151-155.
Cooper, D.M. 1960. Food preferences of larval and adult D. Evolution, 14:41-55.
Cordeiro, A.R., Lewgoy, F., and Tondo, C.V. 1960. Biophysical genetics. IV. Chromatographic patterns of "D. willistoni" races, inter-racial hybrids and the heterosis phenomena. Rev. bras. Biol., 20:69-78.
Cordeiro, A.R., Salzano, F.M., and Marques, V.B. 1960. An interracial hybridization experiment in natural populations of D. willistoni. Heredity, 15:35-45.
Croghan, P.C., and Lockwood, A.P.M. 1960. The composition of the haemolymph of the larva of D.m. J. exp. Biol., 37:339-343.
Crow, J.F. 1960. Genetics Notes. 4th Ed. Minneapolis: Burgess Publ. Co. 149 pp.
1960. Genetics of insecticide resistance: General considerations. Misc. Publ. Ent. Soc. Amer., 2:69-74.
1960. Mutation and selective balance as factors influencing population fitness. Molecular Genetics and Human Disease, Springfield, Ill.: Charles C. Thomas. Chap. 12, pp. 204-211.
Da Cunha, A.B., Dobzhansky, Th., Pavlovsky, O., and Spassky, B. 1959. Genetics of natural populations. XXVIII. Supplementary data on the chromosomal polymorphism in D. willistoni in its relation to the environment. Evolution, 13:389-404.
Da Cunha, A.B. 1960. Chromosomal variation and adaptation in insects. Ann. Rev. Ent., 5:85-110.
D'Amato, F. 1959. Cytological and genetical effects of chemical mutagens. Rep. Second Congr. Eucarpia, Köln, 6 pp.
Daniel, J.C. 1959. Telegony retested. J. Hered., 50:274-298.
Danneel, R., and Eschrich-Zimmermann, B. 1960. Über einen neuen gelben Farbstoff aus den Köpfen rotäugiger D.-Mutanten. Z. f. Natwforsch., 15b:400-402.
David, J. 1959. Etude quantitative du développement de la Drosophile élevée en milieu exenique. Bull. Biol. Fr. Belg., 93:472-505.
1959. Influence de l'âge de la femelle sur les dimensions des oeufs chez D.m. C. R. Acad. Sci., Paris, 249:1145-1147.
Dawood, M.M. 1961. The genetic load in the second chromosomes of some populations of D.m. in Egypt. Genetics, 46:239-246.
De Marinis, F. 1959. The nature of asymmetry and variability in the double Bar-eyeless D. Genetics, 44:1101-1111.
De Marinis, F., and Sheibley, F.E. 1960. Preliminary tests with some synthetic amides on the action of Bar eye D. (Abstr.) Rec. Genet. Soc. Amer., 29:66; and Genetics 45:984.
Demerec, M., and Kaufmann, B.P. 1961. D. Guide. 7th ed. Washington, D.C.: Carnegie Institution of Washington. 47 pp.
Di Pasquale, A. 1960. Il carattere 'macchie brune' in D.m. e la sua risposta ai raggi X. Atti A.G.I., 5:117-126.
Di Pasquale, A., and Santibanez, K.S. 1960. Fecundity in several lines of D. simulans and D.m. Atti A.G.I., 5:93-100.
Doane, W.W. 1960. Completion of meiosis in uninseminated eggs of D.m. Science 132:677-678.
1960. Developmental physiology of a female sterility mutant in D.m. Ph.D. Thesis, Yale Univ.
1960. Developmental physiology of a female sterility mutant in D.m. (Thesis Abstr.) Yale J. Biol. Med., 32:478.
1960. Developmental physiology of the mutant female sterile(2)adipose of D.m.

I. Adult morphology, longevity, egg production, and egg lethality. J. Exp. Zool. 145:1-22.
1960. Developmental physiology. II. Effects of altered environment and residual genome on its expression. J. Exp. Zool., 145:23-42.
Dobzhansky, Th. 1960. Bearing of evolutionary studies of D. on understanding of human evolution. Scientia, 54:4 pp.
1959. Evolution of genes and genes in evolution. Cold Spr. Harb. Symp. Quant. Biol., 24:15-30.
1959. Variation and evolution. Proc. Amer. Phil. Soc. 103:252-263.
Dobzhansky, Th., Krimbas, C., and Krimbas, M.G. 1960. Genetics of natural populations. XXX. Is the genetic load in D. pseudoobscura a mutational or a balanced load? Genetics, 45:741-753.
Dobzhansky, Th., Levene, H., Spassky, B., and Spassky, N. 1959. Release of genetic variability through recombination. III. D. prosaltans. Genetics, 44:75-92.
Dobzhansky, Th., and Pavlovsky, O. 1960. How stable is balanced polymorphism? Proc. Nat. Acad. Sci., U.S., 46:41-47.
Dutt, M.K. 1959. Degradational action of deoxyribonuclease on unfixed chromosomes of D. and grasshoppers. Nucleus, 2:85-98.
Dutt, M.K., and Kaufmann, B.P. 1959. Degradational action of deoxyribonuclease on unfixed chromosomes of D. and grasshoppers. Nucleus, 2:85-98.
Edington, C.W., Epler, J.L., and Regan, J.D. 1960. The frequency-dose relation of Y chromosome suppressed recessive lethals in D. (Abstr.) Rec. Genet. Soc. Amer., 29:67; and Genetics, 45:985.
Ehrman, L. 1960. A genetic constitution frustrating the sexual drive in D. paulistorum. Science, 131:1381-1382.
1960. A preliminary study of mating behavior in D. paulistorum. Amer. Nat., 94:189-191.
1960. The genetics of hybrid sterility in D. paulistorum. Evolution, 14:212-223.
1960. The genetics of hybrid sterility in D. paulistorum. Diss. Abstr., 20:3928.
Ehrman, L., and Strickberger, M.W. 1960. Flies mating: a pictorial record. A species of fruit fly D. paulistorum, occasions some exceptional photos. Nat. Hist., 69:28-33.
Ensign, S.E. 1960. Reproductive isolation between D. tolteca and related species. Evolution, 14:378-385.
1960. Reproductive isolation between D. tolteca and related species. Diss. Abstr. 20:3438.
Erk, F.C. 1960. A study of genetic control over segregation in a translocation heterozygote. (Abstr.) Rec. Genet. Soc. Amer., 29:68; and Genetics, 45:986.
Fahmy, O.G., and Fahmy, M.J. 1959. Complementation among the sub-genic mutants in the r-locus of D.m. Nature, Lond., 184:1927-1929.
1959. Differential gene response to mutagens in D.m. Genetics, 44:1149-1171.
1960. Cytogenetic analysis of the action of carcinogens and tumour inhibitors in D.m. VI. The mutagenic cell stage response of the male germ line to the "nitrogen-mustard" derivatives of amino-acids, carboxylic acids and amines. Genet. Res., Camb., 1:173-188.
1960. Cytogenetic analysis of the action of carcinogens and tumor inhibitors in D.m. VII. Differential induction of visible to lethal mutations by related nitrogen mustards. Genetics, 45:419-438.
1960. Cytogenetic analysis of the action of carcinogens and tumor inhibitors in D.m. VIII. Selective mutagenic activity of S-2-chlorethylcysteine on the spermatogonial stages. Genetics, 45:1191-1203.
1960. Mutagenicity in the sperm of D. and the structure of the "nitrogen-mustard" molecule. Heredity, 15:115-128.
Faulhaber, I. 1959. Biochemische Untersuchungen zum Eiweiss-Stoffwechsel der Letalmutante letal giant larvae (lgl) von D.m. Z. Vererbungsl., 90:299-334.
Forbes, C. 1960. Nonrandom assortment in primary nondisjunction in D.m. Proc. Nat. Acad. Sci., U.S., 46:222-225.
Fourche, J. 1959. La respiration embryonnaire chez D.m. Comparaison de deux souches et de leurs hybrides. C. R. Acad. Sci., Paris, 249:1148-1150.
1959. La respiration embryonnaire chez D.m. Influence de l'âge des parents sur la consommation d'oxygène des oeufs. C. R. Acad. Sci., Paris, 249:1400-1402.
Fox, A.S. 1959. Genetic determination of sex-specific antigens. J. Nat. Cancer Inst. 23:1297-1308.

1959. Problems in genetic control of protein synthesis. (Abstr.) Science, 130: 1417-1418.
Franganillo, A.R. 1959. Estudio genético en poblaciones de D. de Córdoba. Arch. de Zootec., 8:242-276.
Friedman, F., Burton, L., Kaplan, M.L., Kopac, M.J., and Harnly, M.H. 1959. The etiology and development of a melanotic tumor in D. Proc. Conf. Biol. Norm. & Atypical Pigment Cell Growth, 1957, 4:279-299.
Frost, J.N. 1960. The occurrence of partially fertile triploid metafemales in D.m. Proc. Nat. Acad. Sci., U.S., 46:47-51.
1961. Autosomal nondisjunction in males of D.m. Genetics, 46:39-54.
Frydenberg, O., and Sick, K. 1960. The selection of st and cn alleles on different genetical backgrounds in D.m. Hereditas, 46:601-621.
Fujio, Y. 1960. Studies on the development of eye-antennal discs of D.m. in tissue culture. (Abstr.) Jap. J. Genet., 35:257. (In Japanese.)
Fung, S.-T., and Gowen, J.W. 1960. Role of autosome-IV in D.m. sex balance. (Abstr.) Rec. Genet. Soc. Amer., 29:70-71; and Genetics, 45:988-989.
Fuscaldo, K.E., and Jones, H.H. 1959. A method for the reconstruction of three-dimensional models from electron micrographs of serial sections. J. ultrastructure Res., 3:1-10.
Gardner, E.J. 1960. Tumorous head genes in fly populations. Farm & Home Sci. (Utah Sta.), 21:40-41, 57.
Gardner, E.J., and Berseth, W.D. 1960. Population study of isoalleles associated with tumorous head in D.m. (Abstr.) Rec. Genet. Soc. Amer., 29:71; and Genetics, 45:989
Gardner, E.J., Turner, H.J., and Berseth, W.D. 1960. Maternal effect transferred by injection and further analysis of temperature effective period for tumorous head in D.m. Genetics, 45:905-913.
Ghelelovitch, S. 1960. Sur le déterminisme de la sensibilité à l'action tumorigène des rayons X sur D.m. Meig. C.R. Acad. Sci., Paris, 250:1387-1388.
Gibson, J.B., and Thoday, J.M. 1959. Recombinational lethals in a polymorphic population. Nature, Lond., 184:1593-1594.
Gilbert, N. 1960. Quantitative inheritance in D. J. Genet., 57:77-83.
Gill, K.S. 1960. Developmental physiology of five female-sterile mutants in D.M. (Abstr.) Anat. Rec., 138:351.
Glass, B. 1957. Induction of mutations with radiation. Proc. Inter-Amer. Symp. Peaceful Appl. Nuclear Energy, 1:569-576.
1960. The influence of immediate versus delayed mating on the life span of D. In The Biology of Aging: A Symposium (B.L.Strehler, ed.) pp.185-187.
Glassman, E. 1960. Genetic complementation between mutants of D.m. lacking xanthine dehydrogenase. (Abstr.) Rec. Genet. Soc. Amer., 29:71; and Genetics, 45:989.
Glassman, E., and Pinkerton, W. 1960. Complementation at the maroon-like eye-color locus of D.m. Science, 131:1810-1811.
Goldschmidt, E., and Hadorn, E. 1959. Host-transplant interaction in biosynthesis of D. pteridines. J. Embryol. exp. Morph., 7:316-329.
Gottschewski, G.H.M. 1960. Morphogentische Untersuchungen an in vitro wachsenden Augenanlagen von D.m. Roux' Arch. EntwMech. Organ., 152:204-229.
Götz. W. 1959. Rassenbiometrische Studien an Insekten. Arch. Jul. Klaus-Stiftg., 34: 246-252.
Gowen, J.W. 1960. AD. intersex-triploid. Genetics, 45:139-142.
Graf, G.E., Hadorn, E., and Ursprung, H. 1959. Experiments on the isoxanthopterin metabolism in D.m. J. Insect Physiol., 3:120-124.
Green, M.M. 1959. Effect of different wild-type isoalleles on crossing-over in D.m. Nature, Lond., 184:294.
1959. Non-homologous pairing and crossing over in D.m. Genetics, 44:1243-1256.
1959. Putative non-reciprocal crossing over in D.m. Z. Vererbungsl., 90:375-384.
1959. Spatial and functional properties of pseudo-alleles at the white locus in D.m. Heredity, 13:302-315.
1960. A new heterochromatin effect in D.m. Proc. Nat. Acad. Sci., U.S., 46: 524-528.
1960. Apparent double crossing-over in a short genetic interval in D.m. Nature, Lond., 186:990-991.
1960. Double crossing over or gene conversion at the white loci in D.m.? Genetics, 45:15-18.
Greenberg, R., and Crow, J.F. 1960. A comparison of the effect of lethal and

detrimental chroom chromosomes from D. populations. Genetics, 45:1153-1168.
Grell, R.F., and Grell, E.H. 1960. The behavior of nonhomologous chromosomal elements involved in nonrandom assortment in D.m. Proc. Nat. Acad. Sci., U.S., 46:51-57.
Griffen, A.B. 1958. Mammalian cytogenetics and the cancer problem. Ann. N.Y. Acad. Sci., 71:1156-1162.
Guest, W.C. 1960. Cytological studies in the littoralis-montana complex of the virilis species group of the genus D. Diss. Abstr., 20:2975-2976.
Guyénot, E., Thélin, L., and Kiortsis, V. 1959. Action mutagène de faibles doses d'irradiation chez D.m. Schweiz, Gesell. f. Vererbforsch. Jahresber., 34:256-264.
Hadorn, E. 1959. Aendert sich die Phasenspezifität von Letalfaktoren? Arch. Jul. Klaus-Stiftg., 34:234-239.
Hadorn, E., Anders, G., and Ursprung, H. 1959. Kombinate aus teilweise dissoziierten Imaginalscheiben verschiedener Mutanten und Arten von D. J. exp. Zool., 142: 159-175.
Hadorn, E., and Walker, I. 1960. D. und Pseudeucoila. I. Selektionsversuche zur Steigerung der Abwehrreaktion des Wirtes gegen den Parasiten. Rev. Suisse Zool., 67:216-225.
Hannah-Alava, A. 1960. Genetic mosaics. Sci. Amer., 202:118-129.
Hardy, D.E., and Wheeler, M.R. 1960. Paracacoxenus, new genus, with notes on Cacoxenus indagator Loew (Diptera: Drosophilidae). Ann. Ent. Soc. Amer., 53:356-359.
Heed, W.B. 1960. Genetic, cytological and morphological clines in island populations of the cardini species group of D. in the West Indies. (Abstr.) Rec. Genet. Soc. Amer., 29:73; and Genetics, 45:991.
Herskowitz, I.H. 1960. Study guide and workbook for genetics. ix + 274 pp. New York: McGraw-Hill Book Co., Inc.
1960. X-linked recessive lethal mutation rates from D.m. oocytes treated with different dosages and dose-rates of X-rays. (Abstr.) Rec. Genet. Soc. Amer., 29:73; and Genetics, 45:991.
Hertwig, P. 1959. Mutationsforschung in ihrer Bedeutung für die Evolution. Nova Acta Leopoldina, 21:117-145.
Hildreth, P.E. 1960. Quantitative aspects of mating behavior in D. Univ. Calif. Rad. Lab. Publ., 9284:1-28.
Hinton, C.W., and Lucchesi, J.C. 1960. A cytogenetic study of crossing over in inversion heterozygotes of D.m. Genetics, 45:87-94.
Hinton, H.E. 1959. Plastron respiration in the eggs of D. and other flies. Nature, Lond., 184:280-281.
Hiraizumi, Y., and Crow, J.F. 1960. Heterozygous effects on viability, fertility, rate of development, and longevity of D. chromosomes that are lethal when homozygous. Genetics, 45:1071-1083.
Hiraizumi, Y., Sandler, L., and Crow, J.F. 1960. Meiotic drive in natural populations of D.m. III. Populational implications of the Segregation-distorter locus. Evolution, 14:433-444.
Hoenigsberg, H.F. 1960. Sexual behavior: a discussion. Evolution, 14:527-528.
Hoenigsberg, H.F., and Santibanez, S.K. 1959. Courtship elements involved in sensorial discrimination in inbred and outbred D.m. Z. Tierpsych., 16:403-409.
1960. Courtship and sensory preferences in inbred lines of D.m. Evolution, 14:1-7.
1960. Intraspecific sensory discrimination in D. aequinoctialis Dobzhansky and D. prosaltans Duda. Z. für Tierpsychologie, 17:133-140.
1960. Observations on the sexual behavior of D. equinoxialis and D. prosaltans. Amer. Nat., 94:382-384.
Hoenigsberg, H.F., Santibanez, S.K., and Sironi, G.P. 1959. Intraspecific sexual preferences in D. prosaltans Duda and in D. equinoxialis Dobzhansky. Experientia, 15:223.
Hollander, W.F. 1959. The inheritance of red eye color in D. Turtox News, 37:158.
Hollingsworth, M.J. 1960. The morphology of intersexes in D. subobscura. J. exp. Zool., 143:123-151.
Horikawa, M. 1960. Developmental-genetic studies of tissue-cultured eye discs of D.m. II. Effects of the metamorphic hormone (celphalic complex) upon growth and differentiation of eye-antennal discs, and strain differences in relation to the metamorphic hormone. Jap. J. Genet., 35:76-83.
Horikawa, M., and Kuroda, Y. 1959. In vitro cultivation of blood cells of D.m. in a

synthetic medium. Nature, Lond., 184:2017.
Horikawa, M., and Sugahara, T. 1959. Effect of radiation on living cells in tissue cultures. Ann. Rep. Nat. Inst. Genet., Japan, 9:104-105.
1960. Studies on the effects of radiation on living cells in tissue culture. I. Radiosensitivity of various imaginal discs and organs in larvae of D.m. Rad. Res., 12:266-275.
1960. Studies on the effects of radiation on living cells in tissue culture. II. Radiosensitivity of cells isolated from various imaginal discs and organs of larvae of D.m. Rad. Res., 13:825-831.
House, V.L. 1960. An analysis of penetrance and expressivity of fourth vein interruption in the Hairless mutant of D.m. relative to variable levels of modifier action. (Abstr.) Rec. Genet. Soc. Amer., 29:73-74; and Genetics, 45:991-992.
Hubby, J.L. 1959. Studies on pteridine metabolism in D. Diss. Abstr., 20:1977-1978.
Hubby, J.L., and Forrest, H.S. 1960. Studies on the mutant maroon-like in D.m. Genetics, 45:211-214.
Hubby, J.L., and Throckmorton, L.H. 1960. Evolution and pteridine metabolism in the genus D. Proc. Nat. Acad. Sci.,U.S., 46:65-78.
Inouye, I. 1959. Different responses to mitromin exhibited by different stocks of wild-type D.m. Jap. J. Genet., 34:407-417. (Japanese with English summary.)
Ito, L. 1960. The lamellar systems of sytoplasmic membranes in dividing spermatogenic cells of D. virilis. J. Biophys. Biochem. Cytol., 7:433-442.
Ives. P.T. 1959. Chromosomal distribution of mutator- and radiation- induced mutations in D.m. Evolution, 13:526-531.
1959. The mutation rate in D. after high doses of gamma radiation. Proc. Nat. Acad. Sci., U.S., 45:188-192.
1960. The effect of γ-rays on fecundity and mutagenesis in Oregon-R males of D. Int. J. Rad. Biol., 2:54-76.
Ives. P.T., Hopkins, R.P., and Clay, R.A.,(Jr.) 1960. A comprehensive test of the mutagenic effects of combined infrared and γ radiation on successive stages of spermatogenesis in adult D.m. (Abstr.) Rec. Genet. Soc. Amer., 29:75-76; and Genetics, 45:993-994.
Jacobs, M.E. 1960. Influence of light on mating of D.m. Ecology, 41:182-188.
Judd, B.H. 1960. Further analysis of exceptions arising by recombination within the white region in D. (Abstr.) Rec. Genet. Soc. Amer., 29:76-77; and Genetics, 45:994-995.
Kaji, S. 1960. Experimental studies on the developmental mechanism of the Bar eye in D.m. VI. Further investigation on the facet-increasing substances of the Bar-eyed mutant. Mem. Kōnan Univ., Sci. Ser., 4:1-17.
Kang, Y.S., Chung, O.K., and Lee, H.Y. 1959. Studies on the classification and the living conditions of Drosophilidae in Korea (II) . On the unrecorded species of D. flies in Korea. Korean J. Zool., 2:61-65. (In Korean with English summary.)
Kaplan, W.D. 1960. Autoradiographic and genetic studies of tritiated thymidine in the testes of D.m. (Abstr.) Rec. Genet. Soc. Amer., 29:77; and Genetics, 45:995.
Kaplan, W.D., and Sisken, J.E. 1960. Genetic and autoradiographic studies of tritiated thymidine in testes of D.m. Experientia, 16:67-73.
Kaufmann, B.P. 1960. Varying patterns of chromosomal fine structure. The Cell Nucleus, Chapter 5.11 pp. 251-263. London: Butterworth & Co.
Kaufmann, B.P., Gay, H., Dutt, M.K., Bal, A.K., and Buchanan, J. 1959. The nature of the materials of heredity. Yearb. Carnegie Instn. Wash., 58:440-449.
1960. The nature of the materials of heredity. Yearb. Carnegie Instn. Wash., 59:441-453.
Kaufmann, B.P., Gay, H., and McDonald, M.R. 1960. Organizational patterns within chromosomes. Int. Rev. Cytol., 9:77-127.
Kikkawa, H. 1959. Report. Biol. Lab. (Cold Spr. Harb.), 69:60-61.
Kikkawa, H., and Abe, K. 1960. Genetic control of amylase in D.m. Annot. Zool. Japan., 33:14-23.
Kimura, M. 1959. A gene frequency cline determined by selection and migration. Ann. Rep. Nat. Inst. Genet., Japan, 9:84-86.
1959. A maximum principle in the genetical theory of natural selection. Ann. Rep. Nat. Inst. Genet., Japan, 9:83-84.
1959. Inverse approach to the estimation of genetic load disclosed by inbreeding. Ann. Rep. Nat. Inst. Genet., Japan, 9:88-90.
1961. Natural selection as the process of accumulating genetic information in

adaptive evolution. Genet. Res., Cambr., 2:127-140.
King, J.C. 1959. Population studies on embryonic development rates. Biol. Lab. (Cold Spr. Harb.), 69:26-27.
1961. Divergent responses to selection by two populations of D.m. Amer. Nat., 95:7-19.
King, R.C. 1960. Oogenesis in adult D.m. IX. Studies on the cytochemistry and ultrastructure of developing oocytes. Growth, 24:265-323.
1960. The hereditary ovarian tumors of the fes mutant of D.m. (Abstr.) Anat. Rec., 138:361.
King, R.C., and Falk, G.J. 1960. In vitro uptake of uridine-H^3 into developing fruit fly oocytes. J. Biophys. Biochem. Cytol., 8:550-553.
King, R.C., Koch, E. A., and Gassens, G. A. 1961. The effect of temperature upon the hereditary ovarian tumors of the fes mutant of D.m. Growth, 25:45-65.
King, R.C., Sang, J.H., and Leth, C.B. 1961. The hereditary ovarian tumors of the fes mutant of D.m. Exp. Cell Res., 23:108-117.
King, R.C., and Vanoucek, E.G. 1960. Oogenesis in adult D.m. X. Studies on the behavior of the follicle cells. Growth 24:333-338.
Kitagawa, O. 1959. The effects of X-ray irradiation on selection response. Ann. Rep. Nat. Inst. Genet., Japan, 9:76-77.
Knez, F. 1959. D. of the Montgomery Arboretum, Illinois. Trans. Ill. St. Acad. Sci. 52:126-127.
Knight, G.R. 1961. Structural polymorphism in D. subobscura collected from various localities in Scotland. Genet. Res., Cambr., 2:1-9.
Köpf, H. 1958. Deitrag zur Topographie und Histologie neurosekretorischer Zentren bei Drosophila. (II. Larven- und Puppenstadien). Deut. Zool. Gesell. Verhandl., 1957:439-443.
Krimbas, C.B. 1959. Comparison of the concealed variability in D. willistoni with that in D. prosaltans. Genetics, 44:1359-1369.
1960. Synthetic sterility in D. willistoni. Proc. Nat. Acad. Sci., U.S., 46:832-833.
Krivshenko, J. 1959. New evidence for the homology of the short euchromatic elements of the X and Y chromosomes of D. busckii with the microchromosome of D.m. Genetics, 44:1027-1040.
Kroeger, H. 1960. Die Entstehung von Form in morphogenetischen Feld. Naturwissenschaften, 47:148-153.
1960. Hypo- and hyperdevelopment of the male genital apparatus and the Bd-M combination in D.m. J. Morph., 107:227-232.
1960. The induction of new puffing patterns by transplantation of salivary gland nuclei into egg cytoplasma of D. Chromosoma, 11:129-145.
Kroman, R.A., and Parsons, P.A. 1960. Genetic basis of two melanin inhibitors in D.m. Nature, Lond., 186:411-412.
Kummer, H. 1960. Experimentelle Untersuchungen zur Wirking von Fortpflanzungs-Faktoren auf die Lebensdauer von D.m. -Weibchen. Z. f. Vergleichende Physiol., 43:642-679.
Kurokawa, H. 1959. Experiments on sexual isolation between races A and B of D. auraria. Anat. Zool. Japan, 32:220-224.
1960. Sexual isolation among the three races, A, B, and C of D. auraria. Jap. J. Genet., 35:161-166.
Laubscher, F.X., and Malan, G.F. 1960. The estimation of fitness. So. African J. Agr. Sci., 3:171-181.
Lee, T.J. 1959. On a new species, D. clarofinis sp. nov. Korean J. Zool., 2:43-45.
Levengood, W.C., and Shinkle, M.P. 1960. Environmental factors influencing progeny yields in D. Science 132:34-35.
Lewis, H.W. 1960. Genetic control of dopa oxidase activity in D.m. I. Analysis of wild type, sable, suppressor-of-sable, and suppressed sable strains. Genetics, 45:1217-1231.
Lewis, H.W., and Lewis, H.S. 1960. Control of dapa oxidase activity by second chromosome factors in D.m. (Abstr.) Rec. Genet. Soc. Amer., 29:80; and Genetics, 45:998.
1961. Genetic conttol of dopa oxidase activity in D.m. II. Regulating mechanisms and inter- and intra-strain heterogeneity. Proc. Nat. Acad. Sci., U.S., 47:78-86.
Lewontin, R.C., and Cockerham, C.C. 1959. The goodness-of-fit test for detecting natural selection in random mating populations. Evolution, 13:561-564.

Lewontin, R.C., and Kojima, K.-I. 1960. The evolutionary dynamics of complex polymorphisms. Evolution, 14:458-472.

Lindsley, D.L., Edington, C.W., and Von Halle, E.S. 1960. Sex-linked recessive lethals in D. whose expression is suppressed by the Y chromosome. Genetics, 45:1649-1670.

Lints, F.A. 1960. Nucleo-cytoplasmic interactions in D.m. Genetica, 31:188-129.

Lüers, H. 1958. Die Bedeutung strahlender Energie für das Erbgut. Math. Naturwiss. Unter., 11:102-106.

1960. Die moderne Evolutionsgenetik. Biologischen Jahresheft 1960 des Verbandes Deutscher Biologen e. V. Iserlohn, 81-89.

1960. Mutationsversuche mit Megaphen. A'rztl. Forschg. 14:127-128.

Lüers, H., and Bochnig, V. 1958. The failure of raising resistant stocks by permanent DDT treatment with sublethal doses in D.m. Itis, 1:72.

Malogolowkin, C., Carvalho, G.G., and da Paz, M.C. 1960. Interspecific transfer of the "sex-ratio" condition in D. Genetics, 45:1553-1557.

Martin, G.A. 1959. Selection for body weight in D.m. Diss, Abstr., 20:1549.

Martin, G.A., and Bell, A.E. 1960. An experimental check on the accuracy of prediction of response during selection. Biometrical Genetics, pp 178-187. Pergamon Press.

Mather, W.B. 1960. Additions to the D. fauna of Australia. Univ. of Queensland Papers, Dept. Zool., 1:229-239.

Maynard Smith, J. 1959. The rate of ageing in D. subobscura. Ciba symposium on The Lifespan of Animals, pp 269-281.

1960. Continuous, quantized and modal variation. Proc. Roy. Soc. B., 152: 397-409.

Maynard Smith, J., and Sondhi, K.C. 1960. The genetics of a pattern. Genetics, 45:1039-1050.

McMaster-Kaye, R. 1960. The metabolic characteristics of nucleolar, chromosomal, and cytoplasmic ribonucleic acid of D. salivary glands. J. Biophys. Biochem. Cytol., 8:365-378.

Medioni, J. 1959. Les organes de stimulation photique chez D.m. Non-spécificité de cette stimulation relativement aux reactions à la lumière. C. R. Soc. Biol., 153:1845-1848.

1959. Mise en évidence et évaluation d'un "effet de stimulation" du aux ocelles frontaux dan de phototropisme de D.m. Meigen. C. R. Soc. Biol., 153:1587-1590.

Merrell, D.J. 1960. Heterosis in DDT resistant and susceptible populations of D.m. Genetics, 45:573-581.

1960. Mating preferences in D. Evolution, 14:525-526.

Meyer-Taplick, T., and Chen, P.S. 1960. Zur Histologie des Mitteldarms normaler und letaler (lme) Larven von D.m. Rev. suisse Zool., 67:245-257.

Milani, R. 1959. Genetical considerations on insect resistance to insecticides. Genetica Agraria, 10:288-308.

Milkman, R.D. 1960. Potential genetic variability of wild pairs of D.m. Science, 131:225-226.

1960. The genetic basis of natural variation. I. Crossveins in D.m. Genetics, 45:35-48.

1960. The genetic basis of natural variation. II. Analysis of a polygenic system in D.m. Genetics, 45:377-391.

1960. Venation polymorphism and genetic variability in D.m. (reply to paper by Bennett et al). Science, 132:1399-1400.

1961. The genetic basis of natural variation. III. Developmental lability and evolutionary potential. Genetics, 46:25-38.

Millicent, E., and Thoday, J.M. 1960. Gene flow and divergence under disruptive selection. Science, 131:1311-1312.

Milne, A. 1957. Discussion following theories of natural control of insect population. Cold Spr. Harb. Symp. Quant. Biol., 22:268-270.

Mitchell, H.K., Chen, P.S., and Hadorn, E. 1960. Tyrosine phosphate on paper chromatograms of D.m. Experientia, 16:410-411.

Mori, S. 1959. Further study of the population effect on the daily periodic emergence of D. Kyoto. U. Col. Sci. Mem., Ser. B, 26:27-28.

Mori, S., and Yanagishima, S. 1959. Variations of D. in relation to its environment. VII. Does D. change its characters during dark life? Jap. J. Genet., 34:151-161. (In Japanese with English summary.)

1959. Variations of D. in relation to its environment. VII. Does dark life change the characters of D.? 2. Jap. J. Genet., 34:195-200. (In Japanese with English summary.)

Morita, T. 1959. Purine metabolism in D.m. II. Ann. Rep. Nat. Inst. Genet., Japan, 9:22-24.

Moriwaki, D., and Fukatami, A. 1960. Selection for sexual isolation in D.m. (Abstr.) Jap. J. Genet., 35:278. (In Japanese.)

Moriwaki, D., and Ikeda, H. 1960. On 'female-producing female' in D. bifasciata. IV. (Abstr.) Jap. J. Genet., 35:278. (In Japanese.)

Moriwaki, D., and Kitagawa, O. 1960. Analysis of "yellowish strain" in D. bifasciata. (Abstr.) Jap. J. Genet., 35:278. (In Japanese.)

Mourad, A.M., and Mallah, G.S. 1960. Chromosomal polymorphism in Egyptian populations of D.m. Evolution, 14:166-170.

Mukai, T., and Burdick, A.B. 1960. Concerning equilibria of heterotic lethals in random mating populations with particular reference to l(2)55i in D.m. Genetics, 45:1581-1593.

Mukherjee, A.S. 1961. Effect of selection on crossing over in the males of D. ananassae. Amer. Nat., 95:57-59.

Muller, H.J. 1960. The chromosomal basis of the mortality induced by X-rays in D. Int. J. Rad. Biol. (1959 Suppl.):321-325.

Muller, H.J., Carlson, E., and Schalet, A. 1961. Mutation by alteration of the already existing gene. Genetics, 46:213-226.

Muller, H.J., and Meyer, H.U. 1959. Further evidence of the relatively high rate of origination of "invisible" detrimental mutations. (Abstr.) Science, 130:1422.

Muller, H.J., and Zimmering, S. 1960. A sex-linked lethal without evident effect in D. males but partially dominant in females. (Abstr.) Rec. Genet. Soc. Amer., 29:83-84; and Genetics, 45:1001-1002.

Mututani, K. 1958. Studies on the resistance of insects to chemicals. III. Induction of the wing abnormalities by ethyl alcohol and their relation to the alcohol resistance in D.m. J. Osaka City U. Inst. Polytech., Ser. D, Biol., 9:189-194.

Nakajima, Y. 1960. Balanced polymorphism of different gene arrangements due to a terminal inversion, In III L, in D. ananassae. III. (Abstr.) Jap. J. Genet., 35:279. (In Japanese.)

Narise, T., Sakai, K., and Iyama, S. 1960. Mass migrating activity in inbred lines derived from four wild populations of D.m. Ann. Rep. Nat. Inst. Genet., 10: 28-29.

Nawa, S. 1960. Genetical and biochemical studies on the metabolism of pteridines in D.m. --The structure of the yellow pigment. Ann. Rep. Nat. Inst. Genet., 10:104-105.

Nicoletti, B., and Lindsley, D.L. 1960. Translocations between the X and the Y chromosomes of D.m. Genetics, 45:1705-1722.

Nigon, V., and Nonnenmacher, J. 1960. Le métabolisme de la thymidine dans les cellules vitellines de l'ovaire chez D.m. C. R. Acad. Sci., Paris, 251:1583-1584.

Oftedal, P. 1959. Synthesis of deoxyribonucleic acid in adult D. Nature, Lond., 184:1961.

Ogita, Z. 1960. Genetical and biochemical studies on negatively correlated cross-resistance in D.m. II. (Abstr.) Jap J. Genet., 35:282. (In Japanese.)

Ohoba, S. 1960. A relation between the expression of melanotic tumors in D.m. and the composition of culture media. (Abstr.) Jap. J. Genet., 35:281-282. (In Japanese.)

Ohsawa, W., Matutani, K., Tsukuda, H., Mori, S., Yanagishima, S., Sato, Y., and Naka, K. 1958. Variations of D. in relation to the environment. II. Variations of D.m. in the medium containing a sub-lethal dose of copper sulfate. J. Osaka City U. Inst. Polytech., Ser. D, Biol., 9:41-68.

Okada, T. 1959. Interspecific and intraspecific variations of the wing-indices in the genus D., in relation to the wing-lengths (Diptera). Kontyu, 27:21-34.

Okada, E., and Waddington, C.H. 1959. The submicroscopic structure of the D. egg. J. Embryol. Exp. Morph., 7:583-597.

Okada, T. 1960. Allomorphosis of the wing-viens vs. wing-membranes in insects. Nat. Sci. Rep. Ochanomizu Univ., 11:35-50.

1960. Notes on Diastatidae from Japan (Diptera). Kontyû, 28:165-167.

1960. On the Japanese species of the genus Amiota Loew (Diptera, Drosophilidae).

Mushi, 34:89-102.
Okada, T., and Chung, Y.J. 1960. Three species of Drosophilidae from South Korea. Akitu, 9:25-30.
Olenov, Y.M., Galkovskaya, K.F., and Pushnitsyna, A.D. 1960. Materials for describing the effect of ionizing radiation on individual development. Tsitologiia, 1:60-77.
Oliver, C.P. 1959. John Thomas Patterson - A tribute on his 80th birthday. Biological Contributions, Univ. Texas Publ., 5914:8-12.
Oshima, C. 1959. DDT-resistance of D. pseudoobscura. Ann. Rep. Nat. Inst. Genet., Japan, 9:18-19.
1959. DDT and dieldrin (DL) resistance of D.m. Ann. Rep. Nat. Inst. Genet., Japan, 9:20-21.
1960. Genetic variations in DDT-resistance of laboratory strains and natural populations of D.m. Ann. Rep. Nat. Inst. Genet., 10:21-22.
Oshima, C., and Kitagawa, O. 1960. Frequencies of deleterious chromosomes in natural populations of D.m. Ann. Rep. Nat. Inst. Genet., 10:23-25.
1960. Genetic structure of natural populations of D.m. (Abstr.) Jap. J. Genet. 35:285. (In Japanese.)
1960. Heterozygous effects of induced lethals and their persistence in populations. Ann. Rep. Nat. Inst. Genet., 10:129-132.
Oster, I.I. 1959. The genetic basis of X-ray induced somatic damage. Rad. Biol., pp. 268-271.
1959. The spectrum of sensitivity of D. germ cell stages to X irradiation. Rad. Biol., pp. 253-267.
Oster, I.I., and Pooley, E. 1960. A comparison of the mutagenic effects of monofunctional and polyfunctional alkylating agents. (Abstr.) Rec. Genet. Soc. Amer., 29:86-87; and Genetics, 45:1004-1005.
Ostertag, W., and Muller, H.J. 1959. Genetic basis of somatic damage produced by radiation. (Abstr.) Science, 130:1422-1423.
Paik, Y.K. 1960. Genetic variability in Korean populations of D.m. Evolution, 14:293-303.
Painter, T.S. 1959. Some values of endomitosis. Biological Contributions, Univ. Texas Publ., 5914:235-240.
Parker, D.R. 1959. Dominant lethal mutation in irradiated oocytes. Biological Contributions, Univ. Texas Publ., 5914:113-127.
1960. The induction of recessive lethals in D. oocytes. Genetics, 45:135-138.
Parsons, P.A. 1959. Gentotypic-environmental interactions for various temperatures in D.m. Genetics, 44:1325-1333.
1959. Multiplicative gene action in D. linkage data. Genetica, 30:190-200.
1960. Homeostasis of the sex ratio in D. Nature, Lond., 186:411.
Parsons, P.A., and Kroman, R.A. 1960. Melanin inhibitors and the ebony locus in D.m. Heredity, 15:301-314.
Perkins, D.D. 1955. Tetrads and crossing over. J. Cell. Comp. Physiol., 45(Suppl. 2): 119-149.
Petersen, J.A. 1960. Experimental studies on population physiology. I. Respiratory metabolism on Brazilian sibling species of the "willistoni" group of "D." Rev. bras. Biol., 20:205-216.
1960. Studies on the ecology of the genus "D.". I. Collections in two different life zones and seasonal variations in Rio Grande do Sul, Brazil. Rev. bras. Biol., 20:3-16.
Petru, M., and Havlik, B. 1959. Essigfliegen gefährden Hefekulturen. Zentbl. f. Bakt. Parasitenk. Infekkrank. u. Hyg. Abt. I, Orig., 176:157-159.
Pfeiffer, H.H. 1959. Topochemische Versuche an Interchromomeren des Chromosoms 2R der Speicheldrüsenkerne von D. -Larven. Exp. Cell Res., 18:45-50.
Pipkin, S.B. 1959. Sex balance in D.m.: aneuploidy of short regions of chromosome 3, using the triploid method. Biological Contributions, Univ. Texas Publ. 5914: 69-88.
1960. Sex balance in D.m.: aneuploidy of long regions of chromosome 3, using the triploid method. Genetics, 45:1205-1216.
1960. Sex determination. in The McGraw-Hill Encyclopedia of Science and Technology, pp. 227-232.
1960. Sex combs in unbalanced male forms of D.m. (Abstr.) Rec. Genet. Soc. Amer., 29:88; and Genetics, 45:1006.
Pipkin, S.B., and Sullivan, W.N. 1959. A search for genetic change in D.m. exposed to

cosmic radiation at extreme altitude. Aerospace Med., 30:585-598.
Plus, N. 1960. Utilisation des méthodes de séparation des organites cellulaires pour la purification du virus σ de la Drosophile. C. R. Acad. Sci., Paris, 251:1685-1686.
Poulson, D.F., and Sakaguchi, B. 1960. Evidence concerning the nature of the "sex-ratio" agent in D. (Abstr.) Anat. Rec., 138:376.
Prevosti, A. 1960. Cambios en la heterocigosis por inversión cromosómica al variar por selección la longitud del ala en D. subobscura. Genet. Iber., 12:27-41 (In Spanish).
Pritchard, R.H. 1959. Selection for increased recombination in D.m. - a correction. Amer. Nat., 93:142-143.
Ptashne, M. 1960. The behavior of strong and weak centromeres at second anaphase of D.m. Genetics, 45:499-506.
Purdom, C.E. 1960. Mutagenic effects of nitrogen mustard derivatives of azobenzene compounds in D.m. Biochem. Phar., 5:206-218.
Ramel, C. 1960. Studies on interchromosomal pairing and non-disjunction in D.m. Kungl. Fysiografiska Sällskapets Handlignar, N.F. 71(No. 15):52-53.
Ramel, C., and Eiche, A. 1960. Studies on the effect of homozygous and heterozygous w-alleles on longevity and mating preference in D.m. Hereditas, 45:709-716.
Rapoport, I.A. 1960. Right and left morphological assymetry caused by optic isomeres of emetine. Akad. Nauk SSSR. Dok. 130:1355-1358. (In Russian.)
1960. The mutagenic effect of 1,4-bis-diazoacetylbutane. Akad. Nauk SSSR. Dok., 130:1134-1137. (In Russian.)
1960. The reaction of gene proteins with 1,2-dichlorethane. Akad. Nauk SSSR. Dok., 134:1214-1217. (In Russian.)
Rasmuson, M. 1960. Frequency of morphological deviants as a criterion of developmental stability. Hereditas, 46:511-535.
Rasmuson, B., Green, M.M., and Ewertson, G. 1960. Qualitative and quantitative analyses of eye pigments and pteridines in back-mutations of the mutant w^a in D.m. Hereditas, 46:635-650.
Reddi, O.S., and Auerbach, C. 1961. Sensitivity of the D. testes to tri-ethylene melamine (TEM). Genet. Res., Cambr., 2:63-69.
Reeve, E.C.R. 1959. Some genetic experiments with sternopleural asymmetry in D. (Abstr.) Heredity, 13:412.
1960. Some genetic tests on asymmetry of sternopleural chaeta number in D. Genet. Res., Cambr., 1:151-172.
1961. Modifying the sternopleural hair pattern in D. by selection. Genet. Res., Cambr., 2:158.
Rendel, J.M. 1959. Canalisation of the scute phenotype. Evolution, 13:425-439.
1959. Variation and dominance at the scute locus in D.m. Aust. J. Biol. Sci., 12:524-533.
Rendel, J.M., and Sheldon, B.L. 1960. Selection for canalisation of the scute phenotype in D.m. Aust. J. Biol. Sci., 13:36-40.
Rizki, M.T.M. 1960. Intercellular effects of glucosamine-hydrochloride on tumor formation in D.m. (Abstr.) Anat. Rec. 138:378-379.
Rizki, M.T.M. 1960. Melanotic tumor formation in D. J. Morph., 106:147-158.
1960. Pigmented fat cells in a mutant of D.m. Biol. Bull., 119:134-144.
1960. The effects of glucosamine hydrochloride on the development of D.m. Biol. Bull., 118:308-314.
1960. The nature of the autoflourescence of the fat cells of D. (Abstr.) Anat. Rec. 138:378.
Robertson, F.W. 1959. Gene-environment interaction in the growth of D. (Abstr.) Heredity, 13:413.
1959. Gene-environment interaction in relation to the nutrition and G growth of D. Biological Contributions, Univ. Texas Publ., 5914:89-98.
1959. Studies in quantitative inheritance. XIII. Interrelations between genetic behavior and development in the cellular constitution of the D. wing. Genetics, 44:1113-1130.
1960. Inter-relations between genotype, development and ecology in the growth of D. (Abstr.) Heredity, 14:455.
1960. The ecological genetics of growth in D. 1. Body size and developmental time on different diets. Genet. Res., Cambr., 1:288-304.
1960. The ecological genetics of growth in D. 2. Selection for large body size

on different diets. Genet. Res., Cambr., 1:305-318.
Röhrborn, G. 1959. Mutagenitätsuntersuchungen mit 1,4-Dimethylsulphonoxy-1,4-dimethylbutanan D.m. Z. Vererbungsl., 90:457-462.
1960. Chemische Konstitution und mutagene Wirkung. Klassifizierungsversuch chemischer Mutagene. Experientia, 16:523-529. (In German.)
Rokitskii, P.F. 1959. A study of the process of artificial selection under conditions of natural variability and variability experimentally induced by X-ray. Moskov. Obshch. Isp. Prirody. B. Otd. Biol., 64:75-97. (In Russian with English Summary.)
Rudkin, G.T. 1960. The application of ultraviolet absorbence measurements to problems in cell biology. I.R.E. Trans. Med. Electronics, M-E 7:122-129.
Sakaguchi, B., and Poulson, D.F. 1960. Distribution of the "sex-ratio" agent in the tissues of adults of D. willistoni. Ann. Rep. Nat. Inst. Genet., 10:26-27.
1960. Transfer of the "sex-ratio" condition from D. willistoni to D.m. (Abstr.) Anat. Rec., 138:381.
Sandler, L., and Hiraizumi, Y. 1960. Meiotic drive in natural populations of D.m. IV. Instability at the segregation-distorter locus. Genetics, 45:1269-1287.
1960. Meiotic drive in natural populations of D.m. V. On the nature of the SD region. Genetics, 45:1671-1689.
Sävhagen, R. 1960. Relation between X-ray sensitivity and cell stages in males of D.m. Nature, Lond., 188:429-430.
1960. The relation between the rate of induced translocations and treated cell stages in males of D.m. Hereditas, 46:651-667.
Schlager, G. 1959. Fluctuations and gene-environment interaction of pupation site in D.m. M.A. Thesis, Univ. Kansas, Lawrence, Kan., 51 pp.
1960. Controlling fluctuations of pupation site in D. Trans. Kans. Acad. Sci. 63:243-257.
Schneider, I. 1960. Cytological tests of independent assortment among heteromorphic chromosome pairs in D.m. Ohio J. Sci., 60:65-72.
Schnick, S.McA., Mukai, T., and Burdick, A.B. 1960. Heterozygote viability of a second chromosome recessive lethal in D.m. Genetics, 45:315-329.
Schram, A.C. 1960. Studies on the nutrition of D.m. larvae. II. Studies on the inhibition of yeast by p-aminobenzoic acid. Diss. Abstr., 20:2536.
Schultz, J. 1959. Integrative mechanisms in biology. Amer. Nat., 93:209-211.
1959. The role of somatic mutation in neoplastic growth. "Genetics and Cancer", U. of Texas Press, 25-42.
Schultz, J., and Rudkin, G.T. 1960. Direct measurement of deoxyribonucleic acid content of genetic loci in D. (Abstr.) Science, 132:1499-1500.
Seecof, R.L. 1960. The effects of ploidy changes on enzyme activities in D.m. (Abstr.) Rec. Genet. Soc. Amer., 29:92-93; and Genetics, 45:1010-1011.
Seidel, S. 1960. Aufhebung der Sterilität von "Transformer"-Männchen durch Erzeugung von Keimbahn-Mosaiken. Naturwissenschaften, 47:609-610.
Seidel, F., and Buchholtz, Ch. 1960. Die Dosisleistung bei Durchstrahlung biologischer Objekte mit nadelförmigen Bündeln von Röntgenstrahlen. Naturwissenschaften, 47:260-261.
Sengun, A. 1959. Untersuchungen uber die Feinstruktur der Riesenchromosomen in verschiedenen Geweben von D. subobscura Coll. Istanbul. U. Fen Fakul. Mecmuasi, 24B:39-47.
Serra, J.A. 1958. Some apparently "Lysenkoan" aspects of modern genetics. Rev. Port. Zool. Biol. Ger., 1:331-361.
1959. Gene theory: A model of the gene and its sub-units. Nucleus, 2:9-22.
1959. On the nature of overdominance and overdominant alleles. Rev. Port. Biol. Ger., 2:9-36
Shapard, P.B. 1960. A physiological study of the vermilion eye color mutants of D,m. Genetics, 45:359-376.
Shatoury, H.H.El 1959. The development of a pupal lethal mutant in D. Caryologia, 12:104-109.
Shult, E.E., and Lindegren, C.C. 1959. A survey of genetical methodology from Mendelism to tetrad analysis. Can. J. Genet. Cytol., 1:189-201.
Sirlin, J.L., and Jacob. J. 1960. Cell function in the ovary of D. II. Behaviour of RNA. Exp. Cell Res., 20:283-293.
Sirlin, J.L., and Knight, G.R. 1960. Chromosomal syntheses of protein. Exp. Cell Res., 19:210-219.

Smathers, K.M. 1961. The contribution of heterozygosity at certain gene loci to fitness of laboratory populations of D.m. Amer. Nat., 95:27-38.

Smith, K.G. 1960. Drosophilidae (Diptera) occurring at Neston, Cheshire. Ent. Mon. Mag., 96:47.

Sobels, F.H. 1960. Chemical steps involved in the production of mutations and chromosome aberrations by X-irradiation in D. I. The effect of post-treatment with cyanide in relation to dose-rate and oxygen tension. Inst. J. Rad. Biol., 2:68-90.

1960. Effect of post-treatment with cyanide on the induction of mutations by X-rays in D. (Abstr.) Int. J. Rad. Biol, 2:230.

1960. Post-radiation modification of the mutation rate in D. by cyanide. (Abstr.) Acta Physiol. -Pharmacol. Neerland., 9:320-321.

Sokal, R.R. 1959. A morphometric analysis of strains of D.m. differing in DDT-resistance. J. Kansas Ent. Soc., 32:155-172.

Sokal, R.R., Ehrlich, P.R., Hunter, P.E., and Schlager, G. 1960. Some factors affecting pupation site of D. Ann. Ent. Soc. Amer., 53:174-182.

Sokoloff, A. 1959. The habitat-niche of American Nosodendridae. The Coleopterists Bull., 13:97-98.

Sokoloff, A., McMullen, E., Gillies, G., and Hollely, V. 1959. Studies in natural and laboratory populations of D. Biol. Lab. (Cold Spr. Har.), 69:48-50.

Somlo, M., and Fukuhara, H. 1959. Étude de la sensibilité au gaz carbonique et de la fixation du CO_2 chez D.m. Biochim. et Biophys. Acta, 36:221-227.

Sonbati, E.M., and Auerbach, C. 1960. The brood pattern for intragenic and intergenic changes after mustard gas treatment of D.♂♂. Z. Vererbungsl., 91:253-258.

Sonneborn, T.M. 1960. The gene and cell differentiation. Proc. Nat. Acad. Sci., U.S., 46:149-165.

Spassky, B., Spassky, N., Pavlovsky, O., Krimbas, M.G., Krimbas, C., and Dobzhansky, Th. 1960. Genetics of natural populations. XXIX. The magnitude of the genetic load in populations of D. pseudoobscura. Genetics, 45:723-740.

Sprackling, L.E.S. 1960. The chromosome complement of the developing eggs produced by D. parthenogenetica Stalker virgin females. Genetics, 45:243-256.

Stalker, H.D. 1960. Chromosomal polymorphism in D. paramelanica Patterson. Genetics, 45:95-114.

1960. Relationship of meiotic drive and inversion association in D. paramelanica. (Abstr.) Rec. Genet. Soc. Amer., 29:94-95; and Genetics, 45:1012-1013.

1961. The genetic systems modifying meiotic drive in D. paramelanica. Genetics, 46:177-202.

Stern, C. 1959. Use of the term "superfemale". Lancet, Dec. 12:1088.

1960. A mosaic of D. consisting of 1X, 2X and 3X tissue and its probable origin by mitotic non-disjunction. Nature, Lond., 186:179-180.

1960. Dosage compensation-development of a concept and new facts. Canad. J. Genet. and Cytol., 2:105-118.

1960. O. Vogt and the terms "penetrance" and "expressivity". Am. J. Hum. Genet., 12:141.

Stevenson, R., and Carter, T.M. 1959. The D. of Sullivan County, Tennessee. J. Tenn. Acad. Sci., 34:226-228.

Stone, W.S., Guest, W.C., and Wilson, F.D. 1960. The evolutionary implications of the cytological polymorphism and phylogeny of the virilis group of D. Proc. Nat. Acad. Sci., U.S., 46:350-361.

Sturtevant, A.H. 1959. Thomas Hunt Morgan, 1866-1945. Biographical Memoirs, 33:283-325.

Taira, T., and Nawa, S. 1959. Studies on the formation of D. eye pigments. Ann. Rep. Nat. Inst. Genet., Japan, 9:21-22.

Taira, T., and Morita, T. 1960. Effects of X-irradiation on biosynthesis of nucleic acids in D. Ann. Rep. Nat. Inst. Genet., 10:25-26.

Taira, T. 1960. Genetical studies on the red and yellow eye pigments in D. Ann. Rep. Nat. Inst. Genet., 10:102-104.

1961. Enzymatic reduction of the yellow pigment of D. Nature, Lond., 187:231.

Takada, H. 1958. Drosophila survey of Hokkaido. X. Drosophilidae from several localities of Hokkaido. J. Hokkaido U. Facul. Sci., Ser. VI., Zool., 14:120-127.

1960. D. survey of Hokkaido XIII. Some remarkable or rare species of D. from the southern-most area in the Hidaka mountain range. Annot. Zool. Japon., 33:188-195.

Takada, H., and Lee, T.J. 1959. On Mycodrosophila koreana sp. nov. from South Korea. Annot. Zool. Japon., 32:94-96.
Takada, H., and Okada, T. 1960. D. survey of Hokkaido, XI. A new species of D. (Sophophora) from Japan. Annot. Zool. Japon., 33:142-145.
Takada, H., and Toyofuku, Y. 1960. Notes on hibernation of Drosophilidae in Hokkaido. Zool. Mag., (Tokyo) 69:223-232.
Takaya, H., Kaji, S., and Inouye, I. 1959. Deficiency of the compound eye of D.m. showing unusual mode of inheritance. (Abstr.) Jap. J. Genet., 34:311. (In Japanese.)
1960. A new hereditary character of the compound eye of D.m., evoked through nutriments and susceptible to selection. Mem. Kōnan Univ., Sci. Ser., 4:115-155.
1960. Hereditary characters of D.m., evocable through nutriments. I. Several characters evoked through nutriments. (Abstr.) Jap. J. Genet., 35:289. (In Japanese.)
1960. Hereditary characters of D.m., evocable through nutriments. II. Selection of the eye deficiency. (Abstr.) Jap. J. Genet., 35:289. (In Japanese.)
1960. Hereditary characters of D.m., evocable through nutriments. III. Special mode of inheritance. (Abstr.) Jap. J. Genet., 35:289.
Tanaka, E. 1960. A study on the difference of temperature responses in several vegtigial strains of D.m. Jap. J. Genet., 35:222-227. (in Japanese with English summary.)
Tantawy, A.O. 1961. Effects of temperature on productivity and genetic variance of body size in populations of D. pseudoobscura. Genetics, 46:227-238.
Tantawy, A.O., and Mallah, G.S. 1961. Studies on natural populations of D. I. Heat resistance and geographical variation in D.m. and D. simulans. Evolution, 15:1-14.
Tantawy, A.O., and Vetukhiv, M.O. 1960. Effects of size on fecundity, longevity and viability in populations of D. pseudoobscura. Amer. Nat., 94:395-403.
Terzaghi, E., and Knapp, D. 1960. Pattern of chromosome variability in D. pseudoobscura. Evolution, 14:347-350.
Thoday, J.M. 1959. Coupling and repulsion linkages under disruptive and stabilising selection. (Abstr.) Heredity, 13:412-413.
1959. Effects of disruptive selection. Proc. Intern. Congr. Zool., 1958, 15: 127-130.
1960. Effects of disruptive selection. III. Coupling and repulsion. Heredity, 14:35-49.
Thompson, P.E. 1960. Effects of some autosomal inversions on lethal mutation in D.m. Genetics, 45:1567-1580.
1960. The effect of some autosomal inversions on lethal mutation in D.m. Diss. Abstr., 20:2508.
Throckmorton, L.H. 1960. Comparative chromatography of D. species and other species of D. Diss. Abstr., 20:2980.
Tokunaga, C. 1961. The differentiation of a secondary sex comb under the influence of the gene engrailed in D.m. Genetics, 46:157-176.
Toropanova, T.A. 1960. Mutation process in populations. Akad. Nauk SSSR. Dok., 132:460-463. (In Russian.)
Toyofuku, Y. 1959. D. survey of Hokkaido, XII. The salivary gland chromosomes of D. ezoana (a preliminary report). J. Fac. Sci. Hokkaido Univ., Series VI, 14:134-139.
Traut, H. 1960. Über die Abhängigkeit der Rate strahleninduzierter Translokationen und rezessiv geschlechtsgebundener Letalfaktoren vom Stadium der Spermatogenese bei D.m. Z. Vererbungsl., 91:201-205.
1960. Zum Problem der Wirkung von verfüttertem Eisensaccharat auf die durch Röntgenstrahlen induzierte Mutationsrate bei D.m. Z. Vererbungsl., 91:325-332.
Tsai, L.S. 1960. The developmental effects of bw^3 deficiency in D.m. (Abstr.) Rec. Genet. Soc. Amer., 29:97; and Genetics, 45:1015.
Tsukamoto, M. 1960. Metabolic fate of DDT in D.m. II. DDT-resistance and Kelthane-production. Botyu-Kagaku, 25:156-162.
1960. Metabolism of DDT in D.m. (Abstr.) Jap. J. Genet., 35:293. (In Japanese.)
Tirner, J.H., and Gardner, E.J. 1958/59. A possible chemically directed mutation in D.m. (Abstr.) Proc. Utah Acad.Sci. Arts & Let., 36:173-174.
1958/59. Differential viability of D. in response to different concentrations of heavy metal salts added to the culture medium. (Abstr.) Proc. Utah Acad. Sci.

Arts & Let., 36:181.
1960. The effect of copper and iron salts and tryptophan on head abnormalities and melanotic tumors in different stocks of D.m. Genetics, 45:915-924.
Ulrich, H. 1960. Die Beziehung zwischen Strahlendosis und Mutationsrate bei Röntgenbestrahlung von D. -Zygoten. Rev. suisse Zool., 67:287-295.
1960. Ergebnisse strahlengenetischer Untersuchungen an D. -Zygoten. Schriftenreihe des Bundesministers für Atomkernenergie und Wasserwirtschaft. Strahlenschutz, Heft 17, 10-24.
United States Atomic Energy Commission 1960. TID-4041 Genetics Reseach, Division of Biology and Medicine Washington 25, D.C.: Office of Technical Services, 117 pp.
Vetukhiv, M.O., and Beardmore, J.A. 1959. Effect of environment upon the manifestation of heterosis and homeostaais in D. pseudoobscura. Genetics, 44:759-768.
Viscontini, M. 1960. Ptérines et génétique chez D.m. Indus. Chim. Belge, 25:1181-1192.
Volkart, H.D., and Rosin, S. 1959. Lokalisation einer unvollstandig penetranten Mutante von D.m. Arch. Jul. Klaus-Stiftg., 34:229-233.
von Borstel, R.C. 1960. Death of D. embryos by exchanges in the tandem metacentric X chromosome. (Abstr.) Rec. Genet. Soc. Amer., 29:97-98; and Genetics, 45: 1015-1016.
1960. Population control by release of irradiated males. Science, 131:878, 880-882.
1960. Sulla natura della letalità dominante indotta delle radiazioni. Atti Assoc. Genet. Ital., 5:35-50.
von Borstel, R.C., and Rekemeyer, M.L. 1959. Radiation-induced and genetically contrived dominant lethality in Habrobracon and D. Genetics, 44:1053-1074.
Waddington, C.H. 1959. Evolutionary adaptation. Československ. Společnost Zool. Věst., 23:289-306.
1960. Experiments on canalizing selection. Genet. Res., Cambr., 1:140-150.
Waddington, C.H., and Perry, M.M. 1960. The ultra-structure of the developing eye of D. Proc. Roy. Soc. Lond., Ser. B, Biol. Sci., 153:155-178.
Walen, K.H. 1961. Studies of cell lethality of a small deficiency in D.m. Genetics, 46:93-103.
Wallace, B., and Madden, C.V. 1959. The radiation project. Biol. Lab. (Cold Spr. Harb.), 69:25.
Warters, M., and Griffen, A.B. 1959. The centromeres of D. Genetica, 30:152-167.
Wasserman, M. 1960. Cytological and phylogenetic relationships in the repleta group of the genus D. Proc. Nat. Acad. Sci., U.S., 46:842-859.
Watson, G.S., and Caspari, E. 1959. Population genetics of cytoplasmic factors. Biol. Lab. (Cold Spr. Harb.), 69:63-64.
Watson, G.S. 1960. The cytoplasmic "sex-ratio" condition in D. Evolution, 14:256-265.
Weaver, E.C. 1960. Somatic crossing over and its genetic control in D. Genetics, 45:345-357.
Weiss, J.M. 1959. The hereditary determination of individual differences in geotaxis in a population of D.m. Diss. Abstr., 20:1072.
Welshons, W.J., and von Halle, E.S. 1960. Further studies of pseudoallelism at the Notch locus of D. (Abstr.) Rec. Genet. Soc. Amer., 29:99; and Genetics, 45:1017.
Westergaard, M. 1960. Chemical mutagenesis as a tool in macromolecular genetics. Abh. Deutsch. Akad. Wiss. Berlin, Klasse Medizin, pp. 30-44.
Wheeler, M.R. 1960. A new genus and two new species of Neotropical flies (Diptera; Drosophilidae). Ent. News, 71:207-213.
1960. A new subgenus and species of Stegana Meigen (Diptera: Drosophilidae). Proc. Wash. Ent. Soc., 62:109-111.
1960. A new subgenus and two new species of Pseudiastata Coquillett (Diptera; Drosophilidae). Bull. Brooklyn Ent. Soc., 55:67-70.
1960. New species of the quinaria group of D. (Diptera, Drosophilidae). Southwestern Nat., 5:160-164.
1960. Sternite modification in males of the Drosophilidae (Diptera). Ann. Ent. Soc. Amer., 53:133-137.
1960. Three new North American Drosophilidae. Southwestern Nat., 5:89-91.
Williamson, D.L. 1959. Carbon dioxide sensitivity in D. affinis and D. athabasca. Diss. Abstr., 20:1914-1915.
Wilson, B.R. 1960. A study of the chemical components of the egg shell of D.m.

Diss. Abstr., 20:4267.
1960. Some chemical components of the egg shell of D.m. I. Amino Acids. Ann. Ent. Soc. Amer., 53:170-173.
1960. Some chemical components of D.m. egg shell. II. Amino sugars and elements. Ann. Ent. Soc. Amer., 53:732-735.
Wolff, S., and Lindsley, D.L. 1960. Effect of oxygen tension on the induction of apparent XO males in D. Genetics, 45:939-947.
Wolsky, A. 1958. The formation of inter-ocellar bristles in D.m. Proc. Calcutta Zool. Soc. 11:1-7.
Woolf, C.M., and Phelps, L.J. 1960. Chromosomal polymorphism in the tumorous-head strain of D.m. Science, 132:1256-1257.
Wren, J.J., and Mitchell, H.K. 1959. Extraction methods and an investigation of D. lipids. J. Biol. Chem., 234:2823-2828.
Wright, S. 1959. Physiological genetics, ecology of populations, and natural selection. Perspectives Biol. and Med., 3:107-151.
Wright, T.R.F. 1960. The phenogenetics of the embryonic mutant, lethal myospheroid, in D.m. J. exp. Zool., 143:77-100.
Wunder, C.C. 1960. Altered growth of animals after continual centrifugation. Proc. Iowa Acad. Sci., 67:488-494.
Wunder, C.C., Crawfor, C.R., and Herrin, W.F. 1960. Decreased oxygen requirement for growth of fruit fly larvae after continual centrifugation. Proc. Soc. Exp. Biol. & Med., 104:749-751.
1959. Combined influence of gravity and temperature upon growth of fruit fly larvae. Growth, 23:349-357.
Würgler, F.E. 1960. Die Sauerstoffabhängigkeit der Abtötungs- und Mutationsrate bei Röntgenbestrahlung von D. -Zygoten. Rev. suisse Zool., 67:295-302.
Yamada, Y. 1960. Analyses of strains of D.m. selected for abdominal bristles. Ann. Rep. Nat. Inst. Genet., 10:90-92.
Yamada, Y., and Kitagawa, O. 1959. Polygenic mutation, induced by X-rays, in biometrical characters of D.m. Ann. Rep. Nat. Inst. Genet., Japan, 9:75-76.
1960. Doubling dose of polygenic characters. Ann. Rep. Nat. Inst. Genet., Japan, 10:133-134.
Yanagishima, S., and Suzuki, N. 1959. Variations of D. in relation to its environment. VIII. $CuSO_4$ resistance in D.m. I. Zool. Mag., Tokyo, 68:231-237.
1959. Variations of D. in relation to its environment. VIII. $CuSO_4$ resistance in D.m. II. Zool. Mag., Tokyo, 68:419-425. (In Japanese with English summary.)
Yoshida, Y.H. 1960. Studies on dominant lethals induced in D. by X-rays. (Abstr.) Jap. J. Genet., 35:296. (In Japanese.)
Zalokar, M. 1960. Sites of ribonucleic acid and protein synthesis in D. Exp. Cell Res., 19:184-186.
Ziegler, I. 1960. Tetrahydrobiopterin-Derival als lichtempfindliche Verbindung Bei Amphibien und Insekten. Z. Naturforsch., 15b:460-465.
1960. Zur Feinstruktur der Augengranula bei D.m. Z. Vererbungsl., 91:206-209.
Zimmering, S. 1959. Modification of abnormal genetic ratios. (Abstr.) Science, 130:1426.
1960. Modification of abnormal gametic ratios in D. I. Evidence for an influence of Y chromosomes and major autosomes on gametic ratios from Bar-Stone translocation males. Genetics, 45:1253-1268.
Zimmering, S., Oster, I.I., and Muller, H.J. 1960. High effectiveness of fast neutrons in inducing minute deletions. (Abstr.) Science, 131:1322.

In Press

Barigozzi, C., and Halfer, C. Anomalie di trasmissione di tumori melanotici studiate col trapianto degli ovari. Atti VI Convegno A.G.I., Roma, 1960.
Barigozzi, C., and Sgorbati, G. Comportamento dei cromosomi CyL e Pm in eterozigosi con cromosoma tu Be. Atti VI Convegno A.G.I., Roma, 1960.
Becker, H.J. 1960. Untersuchungen zur Wirjung des Heterochromatins auf die Genmanifestierung bei D.m. Zool. Anz., Suppl. 24.
Belitz, H.J. Die Anwendung statistischer Methoden in der Biologie. Der Mathematikunterricht, Heft 3/60, 83-103.

Brncic, D. Non-random association of inversions in D. pavani. Genetics.
Carfagna, M., Solima, A., and De Capoa, A. 1960. Nota preliminare sulle variazioni di alcuni fattori di idoneità (fitness) in popolazioni di D.m. periodicamente irradiate. Atti della VI riunione dell'A.G.I., Roma.
Castiglioni, M.C., and Rezzonico, R.G. 1960. First results of tissue culture in D. Experientia.
1960. First results of in vitro cultivation of D.m. tissue. Atti VI Convegno A.G.I., Roma.
Clark, A.M. 1960. Modification of genetic response to X-irradiation in D. Proc. 3rd Austral. Conf. Rad. Biol., Sydney. Butterworth.
Counce, S.J. 1961. The analysis of insect embryogenesis. Ann. Rev. Ent., 6.
David. J. 1960. Contribution à l'étude du déterminisme de certains caractères quantitatifs chez l'imago de la Drosophile. Ann. Univ. Lyon.
Etude quantitative du fonctionnement ovarien chez D.m. Meig.
Di Pasquale, A. 1960. Nuovi dati sulla trasmissione del carattere 'macchie brune' (brown spots) in D.m. Influenza dei cromosomi e del citoplasma. Atti VI Convegno A.G.I., Roma.
Doane, W.W. Corpus allatum-complex and ovarian transplantations in the mutant female sterile(2)adipose of D.m. (Abstr.) Anat. Rec., 138.
Fahmy, O.G., and Fahmy, M.J. Cytogenitic analysis of the action of carcinogens and tumour inhibitors in D.m. IX. The cell stage response of the male germ line to the mesyloxy esters. Genetics.
Gottschewski, G.H.M. Genetische und entwicklungsphyiologische Ursachen der Sterilität. 18 pp.
Hoenigsberg, H.F., Gallucci, E., and Giavelli, A. 1960. X-ray genetic damage in O_2,$N_2$and air in mature and immature germ cells of D.m. Experientia.
Hoenigsberg, H.F., Pozzi, L.V., and Sironi, G.P. 1960. The response of D. testis to X irradiation induction of dominant lethals. VI Convegno A.G.I., Roma.
Iconomidis, J., and L'Heritier, P. 1960. Les relations du virus 6 avec son hôte Drosophile. - Etude de la perte spontanée de l'etat stabilisé. Ann. Génét., 2.
Kanehisa, T. A study on the change of some metals in melanotic tumor formation in D. flies. Gann, 51:4.
Effects of indole-acetic acid on tumor formation in D. virilis. Jap. J. Genet.,11.
Flavine, iso-xanthopterine, and metal metabolisms on the formation of tumors in D. Jap. J. Genet., 10.
Kanehisa, T., and Fujita, K. A relation between tumor formation and xanthine dehydrogenase in D.m. Jap. J. Genet.
Histological observations of the hereditary tumor of D. virilis. Jap. J. Genet.,11:
Khishin, A.F. 1960 Formaldehyde induced changes in male and female germ cells of D.m. I. Dominant lethals. Alexandria J. Agric. Res.
1960. Genetical studies on some natural populations of D.m. Alexandria J. Agric. Res.
1960. The induction of mutations by formaldehyde in D. females. Alexandria J. Agric. Res.
1960. The genetics of heat tolerance in D. m. from different localities. Alexandria J. Agric. Res.
Kikkawa, H. Further studies on the genetic control of anylase in D.m. Jap. J. Genet.
Nawa, S. 1960. The structure of the yellow pigment from D. Bull. Che.. Soc. Japan, 33(11)
Nigon, V., and Nonnenmacher, J. 1960. L'incorporation de la thymidine tritiée durant l'ovogenèse de la Drosophile.
Ogita, Z. 1961. Genetical and biochemical studies on negatively correlated cross-resistance in D.m. I. An attempt to reduce and increase insecticide-resistance in D.m. by selection pressure. Botyu-Kagaku, 26.
Poulson, D.F., and Sakaguchi, B. 1961. The nature of the "sex-ratio" agent in D. Science.
Poulson, D.F., and Waterhouse, D.F. 1960. Experimental studies on pole cells and midgut differentiation in Diptera. Austrlian J. Biol. Sci.
Robertson, F. W. 1960. The ecological genetics of growth in D. 3. Growth and competetitive ability of strains selected on different diets. Genet. Res., Cambr., 1.
1961. The ecological genetics of growth in D. 4. The influence of larval nutrition on the manifestation of dominance in adult body size, a synthesis of

genetic and physiological evidence. Genet. Res., Cambr., 2.
Santibanez, S.K., and del Solar, E.O. Courtship and sexual isolation in D. pavani, Brncic and in D. gaucha, Jaeger and Salazno, Evolution.
Savhagen, R. The frequency of X males and induced autosomal crossovers after irradiation of D.m. males in air or commercial nitrogen. Hereditas.
The relation between X-ray sensitivity and stages of development of treated cells in spermio- and spermato-genesis of D.m. Hereditas.
Sobels, F.H. 1960. Chemische Beeinflüssung des Röntgen induzierten Mutationsprozesses bei D. Naturwissenschaften.
The role of oxygen in radiosensitivation by cyanide in D. (Abstr.) Int. J. Rad. Biol.
Spiess, E.B. 1961. Chromosomal fitness changes in experimental populations of D. persimilis from Timberline in the Sierra Nevada.
Taira, T. 1960. A biochemical study on allelism at Henna-locus in D.m. Jap. J. Genet., 35.
1960. Genetical and biochemical studies on the red and yellow eye pigments of D.m. Jap. J. Genet., 36.
Tantawy, A.O. Developmental homeostasis in populations of D. pseudoobscura. Evolution, 15.
Thoday, J.M., and Boam, T.B. Regular responses to selection. I. Description of responses. Genet. Res., Cambr.
Toyofuku, Y. 1961. Chromosomal polymorphism in natural populations of D. immigrans. Jap. J. Genet., 36.
Toyofuku, Y., and Kimura, Y. 1960. Distribution of D. flies and the flora in Taisei-Mura, a southwestern part of Hokkaido. Zool. Mag. (Tokyo), 69.
1961. D. survey of Hokkaido, XIV. Distribution of D. flies in Taisei-Mura, a southwestern part of Hokkaido in relation to the flora. Jap. J. Zool., 12.
Ulrich, H. 1960. Die Dosisabhängigkeit der Mutationsrate bei Röntgenbestrahlung von D. -Zygoten in Luft und Stickstoff. Verhandl. Dt. Zool. Ges. in Bonn.

Angus, D. Drosophila collection from the Territory of Papua-New Guinea.

The following are the results and ecological notes of flies of the immigrans sp. group caught during February 1961. Results:

	rubida (%)	pararubida (%)	setifemur (%)	Total
Port Moresby	479 (60)	169 (21)	153 (19)	801
Bulolo	245 (45)	199 (36)	105 (19)	549
Lae	14 (3)	247 (58)	169 (39)	430
Kavieng	17 (20)	40 (50)	24 (30)	81
Rabaul	428 (18)	632 (26)	1348 (56)	2408
	1183 (28)	1287 (30)	1799 (42)	4269

It was noted that D. pararubida was the dominant species over fermenting cocoa pods and citrus and that D. setifemur was particularly associated with rotting five corners (Averrhoa carambola).

Barbour, Evelyn and S. Zimmering Preliminary analysis of a Y chromosome from nature carrying a mutant allele of bobbed.

In routine experiments to introduce Y chromosomes derived from males caught in nature into a y f double-X female, it was found that in the presence of one of these Y chromosomes, symbolized Y-+17, the double-X females hatched very late and showed an extreme bobbed effect, but proved to be fertile. Similar results were obtained from sc^4-sc^8/Y-+17 males. To test the dosage effect of one vs. two Y-+17 chromosomes, y In49 f car/y sc^4 w sc^8/Y-+17 females were crossed by w^{ec3}/Y-+17 males. Of 194 y sc^4 w sc^8 males recovered, 121 (62%) appeared non-bobbed and 73 (38%) appeared bobbed. It is inferred that the non-bobbed males carried two Y-+17 chromosomes and the bobbed males only one. On this interpretation, the bobbed allele in the Y-+17 chromosome acts as a typical hypomorph, as described for bb by Stern (1929). Experiments were carried out to determine the effect of this modified Y chromosome on secondary non-disjunction. Females of the constitutions y In49 f car/y^2 sc w^a ec/Y-+17 and y In49 f car/y^2 sc w^a ec/Y-Oregon R, and having approximately the same autosomal background, were crossed by Oregon R males. The frequencies of XX-Y segregations, calculated from F_1 female offspring only, were found to be as follows: 40.2% (2577 F_1♀♀) from the former, and 64.9% (1794 F_1 ♀♀) from the latter. The results suggest a possible impaired pairing site in the Y-+17 chromosome. No information is as yet available on disjunction of X and Y in males carrying the Y-+17 chromosome.

Bateman, Angus J. X-ray induced "crossing-over".

Analysis is continuing of "cross-overs" recovered from matings of b pr vg/ + + + ♂♂ to b pr vg ♀♀, over the period 5 to 11 days from irradiation of the ♂. It had earlier been supposed that r_1 recombinants (b, pr or vg) could be point mutations or deletions as well as true cross-overs, but that r_2 recombinants (b pr or pr vg) must represent true cross-overs. The latter assumption is now found to be untrue for two reasons: some r_2 recombinants are lethal when homozygous; and in some samples the r_2 class is larger than the r_1 class. It is concluded that there are at least 4 modes of origin of "cross-overs" during the period under study (which we presume to consist largely of irradiated spermatocytes)

1. Point mutations (r_1)
2. Deletions (r_1)
3. Illegitimate crossing over (r_1 = deletion; r_2 = duplication)
4. True crossing over ($r_1 = r_2$)

Each illegitimate cross-over will yield one deletion and one duplication. But the duplication would be expected to be more viable in the zygote than the deletion, so that the observed yield of r_2 from this source would be more than that of r_1. We have found that on days 5, 6 and 7 r_1 exceeds r_2
on days 8 and 9 r_2 exceeds r_1
on days 10 onwards $r_1 = r_2$

This is interpreted to mean that the commonest modes of formation of "cross-overs" on days 5, 6 and 7 are point mutations and simple deletions, on days 8 and 9 illegitimate crossing over and on later days true crossing over.

Beatty, R. A. and N. S. Sidhu. A note on the occurrence of bulbous testes ends in Crianlarich strain of Drosophila melanogaster.

The testes of the Crianlarich strain of Drosophila melanogaster show two differences from other strains: (1) the ends are bulbous to a greater or lesser extent, (2) the colour varies from normal yellow to nearly white. The former condition is here named bulbous testes and apparently has not been reported before in this species.

Stern (1941) demonstrated the importance of the vas in determining the shape of the testes. He showed that the vasa deferentia from species with non-coiling testes fail to induce coiling in testes which normally have coils, and vice-versa, that the vasa deferentia of "coiling-species" will cause normally uncoiled testes to coil. In coiling species, the testes are uncoiled and ball shaped without any attachment up to 30 hours after puparium formation (Stern 1941). The coiling of testes is a differential growth function due to a growth promoting substance. The interaction between testes and vas gives rise finally to the imaginal form of testes. This sort of development of testes in males is common in the "coiling species" of Drosophila. However, the high frequency of bulbous testes ends in Crianlarich strain, and a low frequency of cases of uncoiled testes (mostly cases of failure of one testis to coil, and remaining ball-like) show that the differential growth function, of the growth promoting substance, fails to complete the growth in the first case and leaves the testes uncoiled in the second case.

It has been noted that there are cases of failure of completion of growth in testes in D. subobscura also. In one male belonging to a natural large strain from Aberdeen, the testis of one side was found to be ball-shaped, instead of having the elongated and tubular shape, characteristically found in the species.

The uncoiled testis lobe sends no spermatozoa into the vesicula seminalis of its side, and the latter is smaller in size than the normal one of the other side, and empty. This vesicula seminalis is seen to be translucent, (being empty), in a permanent stained preparation.

It is believed that the fertility of the males must be affected, in case of occurrence of ball-shaped testes which yield no mature spermatozoa and their seminal vesicles remaining empty. In the case of bulbous testes, probably the fertility remains unaffected. It is still a matter of speculation that the Crianlarich strain of Drosophila melanogaster does not have a low fertility because of the character of bulbous testes ends and some though very low frequency of uncoiled testes. The overall fertility of the strain probably remains unaffected in spite of the occurrence of some half-sterile males (i.e. males with one testis uncoiled).

The colour of testes in Crianlarich strain varies from yellow to creamy white, the colour of the eyes of the flies always being red. The previous literature on the colour of testes shows that the colour of eyes and pigmentation of the tunica externa of testes go together, i.e. if the eyes are dark coloured as in wild type, the testes are yellow in colour, and flies with light eye colour have light colour of testes also, ranging from yellow to nearly white. In the Crianlarich strain, it has been found that the colour of testes varies from yellow to creamy white, in spite of the colour of eyes always remaining dark, i.e. red.

The bulbous testes ends character is inherited as a recessive. The crossing of Renfrew stock and Crianlarich gives all normals in F_1, and nearly $\frac{1}{4}$ bulbous testes ends, in F_2.

We are grateful to Dr. F. W. Robertson for very kindly providing us the stocks of flies used for these studies.

References

Stern, C. 1941. The growth of testes in Drosophila. I. The relation between vas deferens and testis within various species. II. The nature of interspecific differences. J. Expt. Zool. 87: 113-158, 159-180.

Stern, C. and E. Hadorn. 1939. The relation between the colour of testes and vasa efferentia in Drosophila. Genetics, 24: 162-179.

Table to show the higher incidence of bulbous testes ends in Crianlarich strain compared with Oregon.k.

Strain	No. of flies investigated	No. of flies with bulbous testes ends	No. of flies with bulbous testis end on one side only	.No. of flies with Uncoiled testis on one side	No. of flies with normal testis in the population
Crianlarich	46	25	10	5	6
F_1 generation of Renfrew X Crianlarich (C x R) flies	51	-	1	-	50
F_2 (same)	25	7	2	-	16
Or.K. flies	60	6	4	-	50

Brosseau, George E., Jr. The effect of M(2)S10 on the fertility of some compound XY chromosomes.

On two separate occasions an attempt to establish a stock of the constitution: y^2 su-w^a w^a bb: = /y^+Y; Cy/M(2)S10 X $Y^S X \cdot Y^L$, In(1)EN In(1)dl-49, y v f car/y^+Y; Cy/M(2)S10 failed owing to the infertility of the M(2)S10 males, while their non-Minute brothers were normally fertile. A survey of the effect of M(2)S10 on the fertility of several compound XY's of diverse origin and structure was then undertaken. In these tests, the fertility of males of the constitution X·Y/y^+Y; M(2)S10/+ was compared to their X·Y/y^+Y; Cy/+ brothers. These males were crossed individually with 2 yv/yv;bw/bw females from a stock that consistently yields over 95% fertile matings. Twenty males of each genotype were tested. Over 90% of the non-Minute males were fertile in every case. In contrast, all but one of the compounds showed a lowered fertility when M(2)S10 was present. The exception was $Y^S X Y^L \cdot Y^S$ which showed 100% fertility. Only 60-65% of the males that were X·Y^L, X·Y^S or $Y^S X Y^L$· were fertile. For $Y^S X Y^S \cdot Y^L$ and $Y^S X \cdot Y^L$, In(1)EN In(1)dl-49 the value was 35-40% and for $Y^S X \cdot Y^L$, In(1)EN it was only 15%. In all cases of a lowered number of fertile males, the fertility of the fertile males was drastically reduced, single males often yielding only a very few progeny. No generalization concerning the structure of a compound and the effect of M(2)S10 on its fertility is possible. Nor does the amount of Y chromosome material, as expressed by the number of fertility factors sets present, seem to be important. This latter point also argues against a position effect explanation (M(2)S10 is a strong enhancer of variegation). It is likely that M(2)S10 interacts with some, as yet undefined, property of the sensitive compounds. These results suggest that this factor may not be the same in each of the compounds.

Burdette, W. J. Effect of penicillin on mutation rate following irradiation in different concentrations of oxygen.

Previous work indicated diminution in lethal mutation rate in Drosophila melanogaster following irradiation when antibiotics were administered. These studies have been extended to an inquiry into the effect of penicillin on the alteration in pattern of induced mutations in an atmosphere of oxygen. Males of the st sr e^s ro ca; tu 36a strain were irradiated (3000 r) and lethals detected on the X chromosome by appropriate crosses with the sc^{S1} B InS w^a sc^8 stock. The first group was not treated, the second was irradiated, the third was raised on medium containing 20,000 units of penicillin per ml. of medium and irradiated, the fourth group was maintained one minute before and ten minutes during irradiation in an atmosphere of 100 per cent oxygen, and the fifth group was similar to the fourth except it was raised on medium containing penicillin in the same concentration as group three. After irradiation at 20 hours of age, males were mated successively to different virgin females at intervals of two days. Lethals in the progeny of these respective matings representing successive stages of spermatogenesis during irradiation are indicated by the letters A - G and their number and distribution are recorded in table 1 and figure 1.

Striking reduction in mutation rate was found when penicillin was added to the medium both in the groups irradiated in air and in oxygen. In the latter, the effect is apparent in stages B and C, but in the former it is evident throughout spermato-

Table 1

Stage of Spermatogenesis	Control			3000 r			3000 r Penicillin			3000 r O_2			3000 r O_2 Penicillin		
	Total	Lethals	Per Cent	Total	Lethals	Per Cent	Total	Lethals	Per Cent	Total	Lethals	Per Cent	Total	Lethals	Per Cent
A	795	1	0.12	600	30	5.00	570	17	2.98	229	2	0.87	216	1	0.46
B	654	1	0.15	609	40	6.56	560	26	4.64	287	23	8.01	294	12	4.08
C	649	2	0.30	354	23	6.49	446	16	3.58	175	24	13.71	235	20	8.51
D	647	0	0	278	17	6.11	298	11	3.69	209	6	2.87	178	5	2.80
E	551	0	0	405	10	2.63	400	7	1.75	222	2	0.90	185	1	0.54
F	512	0	0	452	11	2.65	365	3	0.92	211	3	1.74	225	2	1.05
G	440	0	0	365	4	1.24	370	2	0.54	161	1	0.75	213	2	1.14
Total	4248	4	0.09	3063	135	4.41	3009	82	2.73	1494	61	4.08	1548	43	2.78

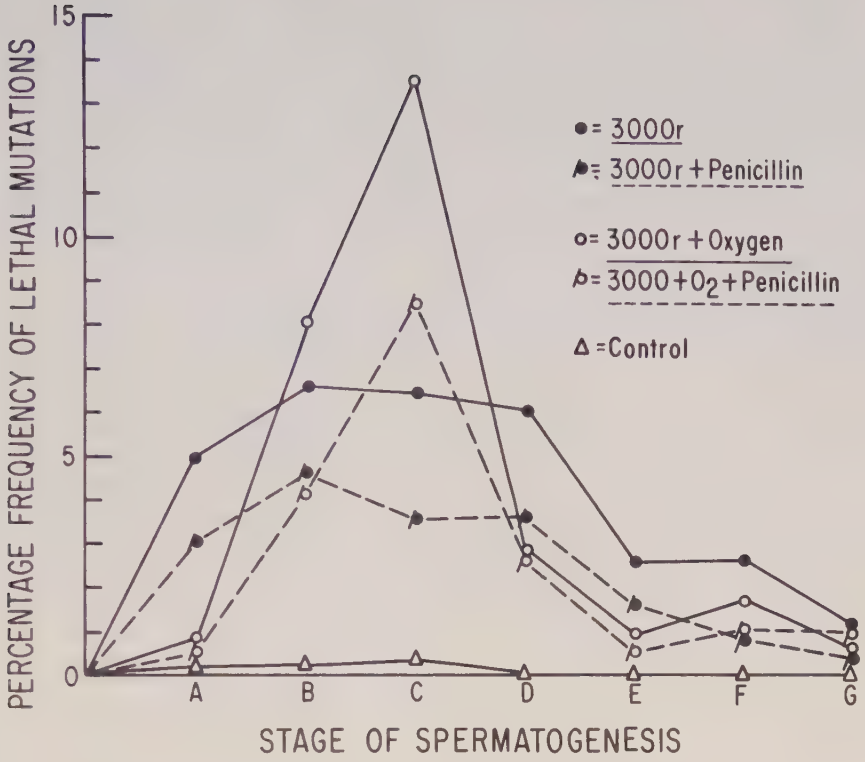


Figure 1: Frequency of Lethal Mutations at Different Stages of Spermatogenesis

genesis. The increase in frequency of lethal mutations when the content of oxygen prior and during irradiation is raised from that in the ambient atmosphere to 100 per cent is greatest in stage C, but this is at the expense of such a change in other stages. When the total frequency of lethal mutations at all stages for the groups irradiated in air is compared to the frequency for those irradiated in 100 per cent oxygen, no increase is found in the latter group. Apparently an increase in percentage of oxygen has resulted in a redistribution of the mutations without increasing total number, whereas penicillin has reduced the number when appropriate groups are compared. The reduction with antibiotic treatment is rather uniform throughout spermatogenesis when irradiation is carried out in air.

Burdick, A. B. l(2)55i at Erie, Pennsylvania.

l(2)55i was found in our so-called "Erie" wild stock (W-1) about a year after the stock had been brought into the laboratory. Various reports by T. Mukai, S. M. Schnick, and myself have dealt with heterozygote viability of this lethal. We have found that this heterozygote is super-viable and have not been able to implicate anything but the single locus itself as a cause of the apparent heterosis. All through these studies we have wondered whether the lethal came from the wild population, or was a mutation that had occurred in the laboratory after the stock had been brought in. Certain considerations have led us to think that even if the lethal had come from the wild population it would not necessarily still be in the population -- six years later. I went back to the Erie area this fall and collected flies again. Our tests show that l(2)55i is still in the Erie wild population.

Carlson, E. A. and R. Sederoff. A selective scheme for recovering pseudoallelic recombinants,

The principle first suggested by Whittinghill (Science 111:377) for the recovery of selected recombinant types has been

"conversion" phenomena, and reverse mutations.

successfully applied in the construction of a lethal selector system for complex loci by Schalet and Chovnick (DIS 34:104). However, this has the inherent disadvantage that four lethals must be used, with each pair of lethals on either side of the allelic region very close to one another. This makes such a system difficult to construct without considerable effort. Furthermore, two sets of such "tester" stocks must be employed so that the wild type crossovers between the alleles can survive in the "sifter" stock used to kill the undesired chromosomes. Finally, this system is valid almost exclusively for single wild type crossovers and it cannot be used to detect double crossovers, reverse mutations, or "conversion" phenomena in the complex locus.

It is possible, however, to use a modification of this principle with certain loci and obtain any wild type event picked up by the "sifter." This proposed system is also simpler to construct and involves only one initial stock for the allelic tests.

In the system illustrated here, the dumpy region is used for the allelic series in question. The stocks are S dp^x Sp / InCyL,Cy and Cy dp^y / InCyL. The "tester" heterozygotes are thus: S dp^x Sp / Cy dp^y ♀♀. In this example the Cy is a dominant visible with recessive lethal manifestation which has been obtained free of its former location near the left end breakpoint of the InCyL,Cy chromosome (Tinderholt, unpublished). S and Sp are also dominant visibles with recessive lethal effects. The alleles dp^x and dp^y represent any two members of the dp series used for analysis. The "sifter" stock has the composition Df(ed^+ dp^+)2MB / S^2 InCyL,Cy. Hence all non-recombinant chromosomes from the "tester" heterozygote are killed by the S^2 InCyL,Cy chromosomes. The distance between S and Cy is about six map units. Hence half of these, carrying neither S nor Cy may be passed on to the "sifter" stock, with a total of 1.5% surviving as a S^+Cy^+dp Sp / S^2 InCyL,Cy combination. The other chromosome in the "sifter" stock, bearing the dumpy deficiency renders the allelic combinations semi-lethal, with a survival to the adult stage of about 5% at 27°C. The total surviving progeny would thus not exceed 7%. Any change occurring in the dumpy region resulting in a non-dumpy phenotype, would complement the deficient area and hence its chances of survival would be nearly unity. Phenotypically there are only two classes of wing mutation in this system -- those with oblique, curly wings and those with very reduced truncated wings. The exceptions would appear as non-dumpy flies. The rate of recombination can be determined by the same procedures outlined by Schalet and Chovnick.

If males are used in this tester stock, and mature sperm are irradiated, then reverse mutations can be selected appearing with an apparent frequency 20 times greater than would be obtained without selective techniques. The tester stock in these reversion studies could use the same scheme as outlined before, but the allele tested would be homozygous. By further increasing the temperature to 29°C, the viability of the heterozygotes is diminished to less than 1% and the selective technique can thus determine reversion frequencies of 1×10^{-7} with the same amount of labor as is presently used for frequencies of 1×10^{-5}.

This system should work for any allelic series which expresses a decrease in viability in compound with a deficiency for its entire region. Other types of selective techniques can be devised using other viability characteristics (such as prune-killer as a "sifter" for pseudoallelism or reverse mutation among prune alleles).
This study is supported by Grant G 14222 from the National Science Foundation.

Chandley, A.C. Mutations induced in presumed spermatocytes

Mating of F_1 males of Drosophila melanogaster following 1000r X-rays has shown maximum sensitivity to the induction of sex-linked lethals and translocations on the 5th day from irradiation followed by a period of low fertility on the 8th. By analogy with the mouse, the 8th day could be expected to represent irradiated spermatogonia and the 5th day irradiated spermatids.

The intervening days 6 and 7 could therefore be expected to represent spermatocytes. These have been investigated for the incidence of dominant lethals, sex-linked recessive lethals, translocations and deleted X's, with the following results:

DAY	5	6	7	8
Dominant Lethal %	38.4	63.0	62.0	59.6
Sex-linked Lethal %	4.08	4.09	4.00	2.62
Translocation %	6.22	5.71	2.92	0.70
Deleted X's %	0.17	0.35	1.24	2.54

Sex-linked lethals and translocations show a rough parallelism with the peak on days 5 and 6. In the case of translocations the level drops sharply through day 7 to the lowest level on day 8.

For sex-linked lethals the high level on days 5 and 6 is maintained through day 7 and then drops sharply to day 8.

Deleted X's (as in previous studies) show a continuous and steep rise from day 5 to a peak on day 8.

Dominant lethals maintain a high level over days 6, 7 and 8. Previously it had been thought that the large percentage of unhatched eggs on day 8 might include some which were unfertilized. However, recent studies on eggs collected within $\frac{1}{2}$ - 1 hr. of laying from matings with day 8 irradiated males have shown almost 100% fertilization - indicated by the presence of polar bodies and early mitotic cleavages.

Amongst the eggs examined were some showing micronuclei, chromosome fragments and stickiness, effects which would lead to breakdown of the mitotic cleavages at an early age and so cause death of the egg.

In order to study directly the effect of X-rays upon spermatocytes, cytological examination was made on testes of irradiated late pre-pupae when only spermatocytes and spermatogonia were present. Twenty-four hours after doses of 1000r and 2000r many of the dividing spermatocytes showed chromosome aberrations including sticky anaphases, chromosome breaks and fragments.

Chandley, Ann C. Timing spermatogenesis in Drosophila melanogaster with tritiated thymidine.

In view of the spate of research on mutation in immature male germ cells it was felt that there was a great need for direct timing of spermatogenesis using tritiated thymidine to label the germ cells. We have injected the abdomen of newly emerged ♂♂ with 0.08 c.mm. of tritiated thymidine (activity 25μC/ml).

On each successive day from injection, the testes of mated and unmated ♂♂ were fixed in 3 : 1 alcohol-acetic, cut at 8 μ and stained with Feulgen. The exposure time for autoradiagraphs was 3 weeks. Labelling of young spermatocytes was detectable on the second day but in the later auxocyte stage the degree of Feulgen staining is so slight and the dispersion of the label so great that it was difficult to recognize label in these cells. We have since found that a much clearer picture of this stage can be obtained using eosin as a counterstain. By day 5, however, groups of very young labelled spermatids were visible, all the labelled nuclei being apparently in a single cyst. With succeeding condensation of the nuclei and agglomeration into sperm bundles, the label became increasingly obvious. To date we have not observed testes more than 8 days from injection. By this time, heavily labelled sperm bundles are present in the testis close to the exit into the seminal vesicle. Assuming that the last stage to incorporate tritiated thymidine is the early spermatocyte the complete life span of a spermatocyte would appear to be 4 days. No differences were apparent between mated and unmated ♂♂ in the rate of spermatogenesis. This study is now being repeated using a heavier dose of tritium, eosin as a counterstain, making autoradiagraphs over a longer period than 8 days, and also looking for labelled sperm in ejaculates.

Divelbiss, J. E. A sterility factor affecting both males and females in Drosophila melanogaster.

An attempt to make a stock of the constitution In(2L)t, Roi·In(2R)Cy, bw^{45a} sp^2 or^{45a}/Ins(2L+2R)Cy, Cy bw^{45a} sp^2 or^{45a} (abbreviated Roi and Cy respectively) failed due to sterility. Outcrosses of Roi/Cy males and females to Oregon-R showed the sterility to be present in both sexes. Since mutant females produced no eggs

and only very few eggs were produced by Oregon-R females mated to mutant males, the observed results are most likely due to sterility rather than zygote lethality. Roi probably arose as the consequence of crossing-over between In(2L)t and In(2L)Cy in In(2L)t, Roi/Ins(2L+2R)Cy, bw^{45a} sp^{2} or^{45a}, hence it would carry the right hand portion of In(2L)Cy. Ives, DIS-25:70, reported the presence of a lethal near each end of In(2L)Cy in Ins(2L+2R)Cy, Cy bw^{45a} sp^{2} or^{45a}. Roi/Cy would be homozygous for the right hand portion of In(2L)Cy and, hence, also for the right hand lethal. However, Roi would carry a duplication for salivary bands 22D2-3; the duplicated piece originating from In(2L)t and carrying l^{+}. This would suggest that the right hand lethal is associated with bands 22D2-3. Roi must also be l^{+} for the left hand lethal. Since the right arms of the two homologues are derived from the same source, they are probably genetically identical. The sterility could be explained by the presence of an undetected mutant in In(2R)Cy which arose previous to the time that Roi was derived, and which would become homozygous in the Roi/Cy heterozygote.

Doane, W. W. Persistence of fs(2)adp in the Kaduna population after four years.

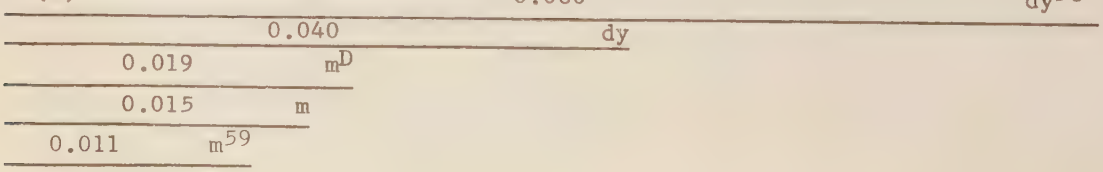
Various traits associated with female sterile(2)adipose and relating to reproductive physiology and fat metabolism suggested that the mutant might persist in the Kaduna wild stock, maintained at the Institute of Animal Genetics in Edinburgh, from which it was originally screened in 1956 (Doane, 1960, J. Exp. Zool., 145: 23-42). In the summer of 1960, 18 sample vials of this stock were received from Dr. A. Robertson of that laboratory and all emerging males were mated individually to Cy/fs(2)adp females. Five non-Cy female progeny from each of these matings were tested for fertility and, where sterility occurred, the lines were perpetuated by crossing their Cy brothers to H-40 females (stock #114 in DIS-34, with dominant markers and cross-over suppressors on chromosomes I, II and III). There followed a breeding program for these lines by means of which the individual Kaduna chromosomes were isolated in the H-40 background so that their effects on the adult fat body and on fertility might be tested. Through this procedure, the factor fs(2)adp has been screened from the descendants of at least 13 of the original 18 samples, suggesting that it persists in the Kaduna wild stock at a fairly high frequency. In addition, other factors affecting the ovaries and fat body have been screened out. Certain second chromosome lethals picked up this way are able, in the heterozygous condition, to mask the effect of fs(2)adp on the fat body. A second chromosome factor, apparently allelic with fs(2)adp but which causes hypertrophy of the fat body without accompanying sterility, is very prevalent in the Kaduna wild stock. This latter mutant is especially well-suited for histochemical studies on fatty tissues.
(This work was supported by a Postdoctoral Fellowship under the Yale University NIH Training Grant in Genetics.)

Dorn, G. L. and A. B. Burdick. Recombination between Df(1)259-4 and various mutants of the miniature-dusky complex in D. melanogaster.

Df(1)259-4 produces a hemizygous effect with the mutants in the miniature but not the dusky cistron. This would seem to indicate that the deficiency extends through the miniature cistron but stops short of the dusky cistron.

Transheterozygotes of Df(1)259-4 with each of three miniature mutants (m, m^{59}, and m^{D}) and two dusky mutants (dy and dy^{58}) were formed. These transheterozygotes have been examined for recombination. All five combinations have been found to yield recombinants.

Below is constructed a genetic map which indicates the relative distances between Df(1)259-4 and each of the five mutants.



If it is assumed that Df(1)259-4 lies to the extreme left, then the gene order agrees with that which we have previously determined (see DIS-33, 1959). Vermilion and garnet markers were also employed in these recombinational experiments. At present, it seems that recombination between Df(1)259-4 and any one of the miniature mutants is also associated with a high negative interference for the outside markers. This does not seem to be the case with the dusky mutants.

Ehrman, Lee. Mutant genes in the Transitional subspecies of D. paulistorum.

Young males from the Bucaramanga, Columbia strain of D. paulistorum were exposed to approximately 4000 r-units of X-rays, and then crossed to untreated virgin females of the same strain. The F_1, F_2, and F_3 progenies were examined for autosomal and sex-linked, dominant and recessive mutations. Although this work is still in progress, and more males will be irradiated, the initial results permit the reporting, for the first time, of mutant genes in this important subspecies:

Delta-Autosomal dominant, lethal when homozygous. This mutation occurred frequently in X-rayed cultures. Wing veins thickened at the margins and the cross-veins. Eyes very rough. This mutation already exists in the Amazonian and the Andean-South Brazilian subspecies of D. paulistorum.

Minute-Autosomal dominant, lethal when homozygous. Bristles reduced in size, especially the scutellar bristles. The developmental period is lengthened, and the viability of both sexes is poor. A Minute exists in the Centro-American subspecies.

Star-Autosomal dominant, lethal when homozygous. This mutation occurred several times in X-rayed cultures. Eyes roughened because of irregularly arranged facets. Star has also been induced in the Amazonian, Andean-South Brazilian, and Centro-American subspecies; thus, it has been acquired in every D. paulistorum subspecies irradiated.

veinless-Sex-linked recessive. This mutation occurred frequently in X-rayed cultures. Many of the wing veins are absent or shortened or interrupted. The wings themselves are warped distally where there are virtually no veins. This mutation was previously induced in the Centro-American subspecies.

The mutations listed above, and others which may be induced and firmly established in stocks, will be employed as genetic markers in the study of reproductive isolating mechanisms (hybrid male sterility and sexual isolation). Because Drosophila paulistorum is now known to represent, at our time level, a number of forms in a state of transition between race and full species, the response of its "bridging" subspecies to a metagenic agent is a necessary prerequisite for further genetic analysis.

(This investigation was supported by a postdoctoral fellowship, GF-9033, from the Division of General Medical Sciences, U.S. Public Health Service.)

Fabergé, A. C., and B. H. Judd. Chromosome breaks by alpha particles.

A fairly high proportion of the chromosome breaks produced by alpha particles in Tradescantia do not rejoin, and by an indirect method, it can be shown that the same is true of the chromosomes of maize endosperm, Fabergé, 1959. For this reason, a preliminary trial was made on treating Drosophila with alpha particles. The penetration of unaccelerated alpha particles is much too low to permit the use of an external source, and the treatments were made by exposing flies to an atmosphere containing Radon, in our case 6.1 microcurie per ml of air. The Radon is believed to equilibrate with the tissues of the fly very rapidly, and since, moreover, the time to gain Radon activity is about the same as the time to lose activity, quite short exposures are practical. The exact distribution, on a microscopic scale, of the ionization does in the tissues of the fly is difficult to assess, since Radon is, in round figures, about 50 times more soluble in lipids than in water. A discussion of these problems will be found in Gray and Read (1942). About 5% of the ionization is also attributable to β-particles, and a negligible amount to gamma radiation.

A solution of Radium bromide in a closed vessel was allowed to equilibrate with its Radon for several weeks. The Radon was then swept over into a one liter spherical flask which has previously been evacuated by admitting air into it which was bubbled through the Ra Br_2 solution. The spherical flask now contained air at atmospheric pressure, mixed with 6.1 μc of Radon per ml. This spherical flask had a small finger-shaped reentrant consisting of an outer sleeve and an inner cylinder,

ground-fitted to each other. The inner cylinder could be put into communication with the atmosphere in the flask by rotating it in the outer sleeve, and aligning several large holes bored through the walls of both cylinder and sleeve. Flies were placed in a lusteroid plastic centrifuge tube, 12.5 mm in diameter, 40 mm long, in which numerous holes had been punched; the stopper of the tube had a cavity filled with mashed banana. To expose the flies this plastic cage was placed inside the re-entrant cylinder, which was then closed to the outside atmosphere, and rotated in its sleeve to align the holes. The Radon atmosphere then diffused among the flies. Since the volume of the re-entrant cylinder was about 10 ml, each exposure resulted in a dilution of the remaining Radon by about 1%. Five exposures of 3.3, 10, 30, 90 and 270 minutes were made, at 25°C, treating about 200 wild type Oregon R males at each exposure. After exposure, the flies were left in fresh food vials in a chemical hood for 30 hours. After this time very little radioactivity remained, and they could be safely handled without special precautions. The males were mated between 48 and 50 hours after exposure to y v f $\frac{C}{XX}$ ♀♀, using 10 ♂♂ and 10 ♀♀ per bottle. Four transfers were made to fresh bottles every 2 days.

The first dose of 3.3 minutes was discarded as having had too little exposure. From the other four doses, a total of 40 duplications were found, among 22922 ♀♀ examined. The relative frequencies of different classes of duplications are about the same as have been observed from X-ray treatment, in so far as can be judged from so small a sample.

X Duplications covering the markers	Radon treatment (all doses) (22922 ♀♀)	X-ray Treatment 1000 r, + 2000 r, + 4000 r (58081 ♀♀) (Bishop 1941)
y	31	298
v	2	0
f	2	1
y v	3	28
y f	2	48
v f	0	4
y v f	0	4

Thus there is no suggestion from this limited information that alpha particles produce duplications of a different nature in Drosophila.

A very small estimate of dominant lethals was also made on a sample of the treated males. The dosage effect curve for dominant lethals from X-rays is of a complex character, and our very small counts do not permit the establishment of the equivalent function for alpha particles. It is thus difficult to make quantitative comparisons of alpha particle and X-ray doses. However, from the dominant lethal counts, as well as the deficiencies, we would hazard the estimate that 1 microcurie per ml of atmosphere for 1 minute is roughly equivalent to 2 r of X-rays, in the region of 1000 r. This estimate may easily be wrong by a factor of 2. Despite the fact that F_1 males were not carefully examined for mutations, several X-linked visibles and autosomal dominants were found. A large fraction were Minutes or Rough-eye mutants. Only 11 of more than 45 mutants were saved for mating. Among the X-linked visibles were y, spl, ec, pn-like, lz-like, sn. Among the autosomal dominants were a furrowed-thorax, a Delta, and several good Minutes.

The method for exposing flies and the apparatus was devised by Dr. D. G. Ott, who, with Dr. W. H. Langham, made preliminary estimates of a reasonable dosage range. We are very much indebted to them for their assistance in carrying out the treatment at the Biomedical laboratory, Los Alamos Scientific Laboratory.

Bishop, Maydelle, 1941 The recovery of a simple and multiple breaks of the X-chromosome of Drosophila melanogaster. Thesis, M. A., University of Texas.

Gray, L. H. and Read, J., 1942 The lethal effect of alpha radiations. IV. The effect of ionizing radiations on the Broad Bean Root. Brit. J. Radiol. 15L 320-336.

Fabergé, A. C., 1959 Production by alpha particles of functionally stable broken chromosome ends in maize. Genetics 44: 279-285.

Fox, Allen S., and Eileen A. Sweeney*. Chemical structure and time of appearance of the sex peptide of males in Drosophila melanogaster.

The presence of a peptide in adult males, but not in females, has been reported previously (Fox, 1956, Physiol. Zool. 24:288; Fox et al., 1959, Science 129:1489) This object, called the "sex peptide", has now been purified by elution from paper chromatograms and subjected to partial chemical characterization.

Acid hydrolysis of the sex peptide yields ten ninhydrin-positive products. These have been identified by paper chromatography of the free products themselves and of their dinitrophenol derivatives, prepared by reaction with 1-fluoro-2,4-dinitrobenzene according to the method of Levy (1954). Eight are conventional amino acids: aspartic acid, glutamic acid, serine, glycine, ∝-alanine, leucine, valine, and methionine. The ninth is ethanolamine. The tenth remains unidentified.

Since ethanolamine lacks a carboxyl group, it must occupy what would otherwise be the C-terminal position of the peptide chain, or constitute a side branch through peptide linkage with one of the dicarboxylic amino acids or phosphate linkage with the serine. Initial attempts to identify the N-terminal residue by preparation of the dinitrophenol derivative of the intact peptide according to the method of Sanger and Thompson (1953), suggest that this position in the peptide is occupied by the unidentified residue. Molar ratios have not yet been determined.

The sex peptide is not present in detectable amounts in male third instar larvae, nor in pupae, nor in males during the first two hours after emergence. In male third instar larvae and pupae, but not in females, there is present instead a substance tentatively identified as phosphoethanolamine. This disappears in newly emerged males just prior to the appearance of the sex peptide.

(Supported by grant C-2440, National Institutes of Health, U. S. Public Health Service, and by a grant from the Rackham Foundation. *National Science Foundation Undergraduate Research Participant in the Department of Agricultural Chemistry and the Honors College of Michigan State University.)

Frost, J. N. Double fertilization mosaics.

In experiments involving triploid females and in which approximately 109,000 offspring were examined eight double fertilization mosaics occurred. In all these experiments the parental origins of the sex chromosomes could be determined. Four of the mosaics were diploid female-intersexes, one was a diploid male-intersex, one was a diploid male-triploid female, one was an intersex-triploid female, and the last was intersexual in both portions of the mosaic. In four of the mosaics the two original egg nuclei had carried complementary chromosome sets, in another the two egg nuclei had carried identical chromosome sets. In three of the mosaics the chromosome sets of the two egg nuclei had been neither complementary nor identical, a fact suggesting that at least some, and perhaps most of the double nuclei in the eggs had arisen from independent meiotic divisions of the two nuclei in a binucleate oogonium.

The distribution and proportions of the mosaic parts were quite variable, only two mosaics being bilateral. Each part of the sex mosaics showed complete autonomy in development.

Frost, J. N. Two mosaics of unusual origin.

A diploid female-intersex mosaic occurred in the following cross: y w 3N (free X) females by y:CY/Gla;D/Sb males. The diploid female portion was yellow, Curly, Glazed, Dichaete, and Stubble and thus (with the possible exception of one X chromosome) obtained all its chromosomes from the male. The intersex portion was yellow, Glazed, and Stubble and arose from a normal zygotic nucleus while the diploid female portion apparently originated from the independent development of a diploid sperm nucleus. The latter could have been produced by a tetraploid spermatogonium.

Another unusual mosaic occurred in a cross of y w (attached-X) 3N females by $y,sc^{8}.Y;L,sp/L,sp;Sb,e/e^{s}$ males. The entire fly was a diploid female and both the left and right sides were yellow, Lobe, Stubble, and ebony, indicating that both of the third chromosomes on each side had come from the male. In addition the left side of the fly was speck and the left eye was completely absent indicating that the left side had received both of its second chromosomes as well as both of its third chromosomes from the male. A satisfactory explanation for this mosaic has not yet been devised.

Frye, Sara H. Evidence that achaete may not be to the right of yellow.

Out of 547 transmissible X-ray-induced "yellow" mutants in scute-8 chromosomes the frequencies of different combinations of affected loci in order of decreasing frequency were as follows: (using the same symbolism as before, Frye, 1960, DIS 34) - - - + (398), - - - - (76), + - - + (45), + - + + (23), + - - - (5). No "yellows" were recovered of the other 3 possible classes (+ - + -, - - + + or - - + -). This obviously means that ac is closer to y than 1J1 and that ac is closer to 1J1 than to bb. None of the foregoing tabulations yet allows for the decision as to whether ac is to the left or to the right of y. However, other evidence suggests that ac may not be to the right of y since genetic analysis of 10 cases of dark yellows, included in the above tabulation, showed that 4 were not deficient or affected at the loci of 1J1, ac or bb (+ $\pm$ + + where $\pm$ represents the dark yellow and the order is assumed to be 1J1 y ac bb), 2 were deficient or affected at the locus of ac, but not 1J1 or bb (+ $\pm$ - +), and 4 were deficient or affected at the loci of 1J1, ac, but not bb. (- $\pm$ - +). This can be seen by comparing the two possible orders (ac to the right and ac to the left of y) of the 4 loci concerned with the 3 recoverable classes of dark yellows.

1J1	y	ac	bb		1J1	ac	y	bb
+	$\pm$	+	+	(4)	+	+	$\pm$	+
+	$\pm$	-	+	(2)	+	-	$\pm$	+
-	$\pm$	-	+	(4)	-	-	$\pm$	+

The latter order (ac to the left of y) is the only one that allows for one of the breaks (in this case, the right one) to fall in a common region. One must also allow for the difference in the size of the regions -- on the basis of breakage the third region is about 6X the first region (Frye, unpublished) regardless of the order of the loci. Another advantage of the suggested order (that of the right hand column) is that it allows for a plus allele of bobbed to border the y locus since those cases of dark yellows that have a normal allele bordering on either the left or the right side of the yellow locus can be explained by position effect, sub-gene deficiency of "point" mutation, whereas those cases where the dark yellows occur between 2 affected loci (as in the last row on the former order) are less easily explained (except as skipping effects on intermediate loci in cases of minute inversions).

Traditional evidence that places ac to the right of y is based chiefly on the mutant sc3, found by Dubinin to affect both achaete and scute strongly, whereas here are 4 cases in which 1J1 and ac were strongly affected but y (which on Dubinin's view would be between them) was only slightly affected.

(Work supported by a grant to H. J. Muller and associates from the U. S. Atomic Energy Commission (Contract AT (11-1)-195).)

Frye, Sara H. Frequency of "transmissible mutation" at the w and f locus in the scute-8 chromosome in relation to X-ray dose in Drosophila.

Males of composition sc^8 B (0-24 hrs. old) were given 500r, 1000r, 3000r or 4000r and these and simultaneous controls were mated to y w In49 f virgin-females (48-72 hrs. old). The offspring recovered were from sperm ejaculated from 1 to 4 days after irradiation. The number of female and male parents introduced per bottle, representing different X-ray doses, were respectively: 500r - 6 prs., 1000r - 8 prs., 3000r - 14 prs., 4000r - 20 prs. Controls were run with 2, 3, 4, 5, or 6 prs. in order to test for crowding. Usually 4 prs. were used. The frequencies of exceptional phenotypes that were transmissible after progeny testing among the F_1 Bar females were for the control, 500r, 1000r, 3000r, and 4000r treatments, respectively: "white" 0.0002% (1/412,439), 0.0050% (14/277,667), 0.0060% (11/180,942), 0.0173% (8/46,067), 0.0217% (9/41,310); "forked" 0.0% (0/263,694), 0.0028% (8/277,667), 0.0033% (6/180,942), 0.0195% (9/46,067), 0.0121% (5/41,310).

Since the number of transmissible mutants per each X-ray dose is small and a genetic analysis was not conducted to determine their qualitative composition no conclusions are drawn (even though most of the w and f mutants were male-viable it does not follow that these are gene mutations since presumptive point mutations,

inversions, or extremely small deletions could survive in the male) concerning their relation to X-ray dose but only their frequencies are reported.
(Work supported by a grant to H. J. Muller and associates from the U. S. Atomic Energy Commission (Contract AT (11-1)-195).)

Frye, Sara H. Persistence of qualitatively diverse "yellow" mutants in scute-8 chromosomes in the absence of selection for one year.

Many "yellow" mutants were induced by exposing young ♂♂'s carrying the scute-8 chromosomes to various X-ray doses, mating them to y w In49 f virgin-females, and recovering the yellow mutants among the F_1 Bar ♀♀'s. After an extensive genetic analysis (Frye, 1958) to determine their qualitative composition each yellow mutant was kept for several years in an unbalanced stock ($y^- sc^8$ B/y w In49 f♀ x y w In49 f♂) which meant that frequent selection was necessary in order to maintain the $y^- sc^8$ B chromosome and to prevent the stock from becoming homozygous for y s In49 f ♀♀'s. However, the last year before these yellow mutant stocks (each stock was kept in a group of 4 vials) were discarded no selection was performed. At the time of discarding (total no. of yellow mutant stocks remaining was roughly 150) I randomly sampled 52 of these stocks to see what qualitative types had persisted and which had not in spite of the absence of selection. All one had to do was to etherize and check for y B ♀♀'s (where they were present no counts were taken to compare the no. of y B ♀♀'s with the no. of y w In49 f ♀♀'s). Out of 30 stocks, selected randomly, the following qualitative types had persisted (using the same symbolism as before, Frye, 1960, DIS 34) - - - + (9), + - - + (7), + - + + (5), - - - - (4), - - - + 1 (3), - - - - 1 (2). Out of 22 stocks the following had not persisted - - - + (3), + - - + (3), - - - - (6), - - - + 1 (2), - - - - 1 (5), + - - + 1 (2), IV$^+$ - - - - (1).

It can be seen that several of the same qualitative types are common to those that did and those that did not persist, and that there is no simple correlation between sheer number of loci absent or affected and the ability to persist, therefore persistence must be a very complex phenomenon. (This is not to be taken as meaning that persistence is independent of qualitative composition of these mutants). It would be of interest to see if one could correlate the no. of generations that is required for different genetically analyzed mutants to become eliminated in a population with their qualitative structure.

Frye, Sara H. Spontaneous "yellows" as gross rearrangements in Drosophila.

Frye (1958) reported 4 yellows, recovered in the female, that arose spontaneously and singly in separate control series in crosses of scute-8 B males to y w In49 f virgin-females, to be attached-X's with one break having occurred in the paternal-X and the other in the maternal-X. Their resemblance to Sidky-like rearrangements (X-ray induced break in one chromosome, spontaneous break in another non-homologous chromosome) is only superficial since in my cases both breaks are spontaneous. Genetic analysis showed that all 4 were deficient or affected at the loci of lJl, y, and ac in the paternal scute-8 chromosome.

In order to see if these 4 yellows acted as if they were structurally the same (as implied by the genetic analysis) 5 virgin-females of each of the 4 yellow stocks were crossed with 5 y w In49 f males in half-pint bottles and a count of the sex ratio was made to see if the proportion of F_1 males to females varied among the 4 yellow stocks; the idea being that if the break in the paternal-X had resulted in more of the X-chromosome being lost or affected in some cases than in others this differential viability would shift the sex ratio in favor of the males. The 4 yellow stocks were designated y^{0001}, y^{0002}, y^{0003} and y^{0004}, and the results (giving the count of the males first) were for each of the above, respectively: 1652 - 730 appx. 2:1; 838 - 684 appx. 1:1; 993 - 727 appx. 1:1; 1146 - 399 appx. 3:1. Thus y^{0001} and y^{0004} are of lower viability than y^{0002} and y^{0003} and perhaps involve a greater loss of distal X-chromosome material (especially the bb$^+$ locus which could not be tested for directly since the attached-X "yellows" carried a Y-chromosome).

The most unexpected fact is that no yellows with attached-X's occurred in the treated series even though all 607 X-ray induced yellows (in scute-8 chromosomes) were progeny tested and analyzed in exactly the same way as the yellows of spontaneous origin. The total number of F_1 females in the treated series here was 583,248 and that in the above-mentioned controls was 412,439.

Also, there is no reason to conclude that "yellows" occurring spontaneously in the scute-8 chromosome are exclusively or even highly likely to always be exchanges with Y^S, but may be due to breaks the results of which may be classifiable as minute chromosome changes, crossovers, or gross rearrangements. (None of the above 4 involved exchanges with Y^S. and out of a total of 10 "yellows" arising spontaneously in the scute-8 chromosome of males two were found to involve an exchange with the Y^S. Tests for the exchange with Y^L were not made.)

Other spontaneous gross rearrangements involving the tip of the X-chromosome are known (Burkart, 1930 - Blond, Muller, 1943 - "double-X") and the tip of the autosomes (Bridges, 1919 - Pale). Chromosome tips may be more likely to enter into gross rearrangements spontaneously (and possibly when X-rayed) than is ordinarily thought.

Fuscaldo, Kathryn E., and Allen S. Fox. Immunogenetic studies of white-variegated position effects.

Agar-diffusion techniques were employed to investigate the antigenic specificities of proteins extracted from the following stocks of D. melanogaster: $In(1)w^{m4}$; a derivative, $In(1)w^{m4w}$, in which the rearrangement is unaltered but a change has occurred in the white pseudoallelic segment (Schultz, 1943, D.I.S. 17:64); the translocation, $T(1:4)w^{m5}$; the mutants w, w^e, and w^{a2}; the double mutant, bw cn; and the isogenic wild stock, Oregon-R-I.

In all cases an alteration of the relationship of heterochromatin to the white pseudoallelic segment resulted in a change in the immunochemical properties of an antigen, designated H(w)-1. The protein H(w)-1 exhibits a higher antibody combining power in the inversion and translocation stocks than in the wild stock. The difference most probably is associated with a difference in the number of combining sites on the antigen molecule, along with a small difference in the configuration of the antigenic site. The effect is reminiscent of the effect of the Y chromosome on the antigen Y-1 (Fox, 1959, J. Nat. Cancer Inst. 23:1297).

The properties of H(w)-1 extracted from the mutants w, w^e, and bw cn are the same as that extracted from the wild stock. H(w)-1 extracted from w^{a2}, on the other hand, behaves like that extracted from the inversion and translocation stocks. The mutants w and w^e occupy a locus to the right of that occupied by w^{a2} (Lewis, Green). It thus appears that some, but not all, alterations of the white pseudoallelic segment affect the structure of H(w)-1. Furthermore, the effect is not directly associated with eye pigmentation (vide bw cn).

The results may be rationalized by the hypothesis that the euchromatic white pseudoallelic segment determines the primary structure (amino acid sequence) of the protein H(w)-1, but that the tertiary structure of the protein depends on the relationship of this euchromatic segment to heterochromatin. The participation of heterochromatin in the determination of tertiary structure has been postulated previously in connection with the effects of the Y chromosome on the protein Y-1, and the respective roles of euchromatin and heterochromatin in protein synthesis have also been discussed (Fox, 1959, Science 130:1417).

(Supported by grant C-2440 from the National Institutes of Health, U. S. Public Health Service.)

Ghini, Clara. Effect of nebularine and EOC (8-etoxy caffeine) on selection response for sternopleural hairs in D. melanogaster.

The effects of two mutagens: EOC(8-etoxy caffeine) and nebularine on the induction of genetic variability as shown by progress under selection, has been studied. An isogenic stock has been used; the character selected for was high number of sternopleural hairs. A family selection method was applied every two generations. The mutagens were given by intra-abdominal injections to adult flies of both sexes, aged 18-24 hours. Three concentrations (.050%; .025%; .010%) were tested for nebularine and two for EOC (.30%; .10%)

With nebularine a positive response to selection was obtained from the first generation on, but the response continued only in the .025% lines. From average number of hairs of 20.31 $\pm$ .099, after 8 selection cycles corresponding to 16 generations, one obtained values of 26.01 $\pm$ .379 and 29.52 $\pm$ .302 in two different replications. In the same period the two untreated lines from an initial average value of 20.74 $\pm$ .098 reached a level of 21.29 $\pm$ .128 and 20.96 $\pm$ .106. The response to selection was found associated with an increase in variability, expressed both in terms of standard deviations and of coefficients of variation. With EOC the average

values of sternopleural hairs reached, after 8 selection cycles, were 21.22±.115 and 21.19±.109, not significantly different from the values reached by untreated control lines. From experiments with plants one knows the mechanism of action of the two mutagens as being very different, EOC causing mainly chromosome breakage while nebularine (Ehrenberg and Gustafson, 1954) is supposed to produce mainly point mutations. Experiments designed to compare the mutagenic action of these substances in D. melanogaster are in progress.

Grell, R. F. The penetrance of sparkling-Cataract.

A research note in DIS 33: 150 (1959) by H. J. Muller states that a single fourth chromosome carrying sparkling-Cataract (spa^{Cat}) in combination with two normal fours produces a wild-type eye. Flies have been synthesized in this laboratory that carry three free fourth chromosomes, each marked with a single dominant mutant ($spa^{Cat}/ci^{D}/ey^{D}$), in an otherwise diploid background. The penetrance of spa^{Cat} in these triplo-four flies is complete, although its expression is less extreme than is one dose of spa^{Cat} in the diplo-four condition.

The mutant, spa^{Cat}, has also been used to mark the free fourth chromosome of triplo-four females that are homozygous for T(3;4)86D in order to follow the assortment of the free four and an extra Y chromosome. In this situation one dose of spa^{Cat} was found to be fully penetrant and classifiable both in the mother and in her triplo-four progeny. Mothers of this genotype ($T(3;4)86D/T(3;4)86D/spa^{Cat}$) mated to diplo-four males carrying two normal fours produced 332 spa^{Cat} and 329 non-spa^{Cat} offspring [Genetics 44: 421 (1959)]. Mothers of the same genotype mated to $M\text{-}4/ey^{D}$ males produced non-eyeless-Dominant flies that were either M-4 or spa^{Cat}, clearly demonstrating that the mutant phenotype is always classifiable when present in one dose in triplo-four flies.

It is of interest that one dose of spa^{Cat} in the F_1 diplo-four hybrid between D. melanogaster and D. simulans (synthesized by E. H. Grell) shows an extreme mutant phenotype, whereas spa^{Cat}/simulans-four in an otherwise completely melanogaster background, as observed by Muller, is wild type.

(This work was done at the Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee, operated by Union Carbide Corporation for the United States Atomic Energy Commission.)

Hildreth, P. E. and J. C. Lucchesi. Fertilization in D. melanogaster and D. virilis.

Early findings by Huettner (J. Morph. 39:249, 1924) concerning the occurrence of polyspermy in D. melanogaster have been supported by Counce (DIS 33:127, 1959) and extended to eight additional species, including D. virilis; this author found an average of 5 to 6 spermatozoa in D. melanogaster and 50 to 100 spermatozoa in D. virilis eggs.

Using the Feulgen whole-mount procedure of von Borstel and Lindsley (Stain Technol. 34:23, 1959), preliminary work on D. melanogaster eggs failed to show polyspermy (Hildreth, unpublished); therefore further cytological examination of D. melanogaster and also D. virilis eggs was conducted. In D. melanogaster 96 eggs were found in meiotic stages; among these 91 had a single sperm, 2 had two sperms, and 3 had no visible sperm. This is consistent with the observations of Hinton and Lucchesi (Genetics 45:87, 1960). Among 127 meiotic eggs of D. virilis, 87 eggs had a single sperm, no sperm was visible in 40 eggs, and no case of polyspermy was observed.

The reason for the differences between our results and those of Huettner and Counce are not known. Autoradiographic studies of fertilization in D. virilis are now being conducted in an attempt to obtain further information on the question of polyspermy.

(This work was carried out under the auspices of the U.S. Atomic Energy Commission.)

Hochman, B. On the viability of the brown-Variegated^{1}/brown-Variegated^{57e} heterozygote.

More than 20 brown-Variegated (bw^{V}) alleles are listed in Bridges and Brehme (1944). Their viabilities in the homozygous state are described variously as, generally lethal, nearly always lethal, lethal in 95% of the cases, etc. The first one found,

bw^{V1} (usually referred to as Plum (Pm)), is reported to be, "generally lethal when homozygous, and also lethal with all other brown-Variegateds."

During the course of an experiment involving the Notch locus, data were obtained on the viability of the Pm/bw^{V57e} genotype which demonstrate that the statement in the preceding sentence does not apply to this combination of bw^{V} alleles. (Dr. E. H. Grell produced bw^{B57e} by irradiating the SM1 chromosome. The presence of Cy in SM1 (see DIS 27: 57-58) precludes an examination of the bw^{V57e} homozygote. When heterozygous with wild type, bw^{V57e} causes the same eye mottling as Pm/+. Under certain genetical and environmental conditions, it was observed that 0.2-0.4 of expected Pm/bw^{V57e} individuals reached the adult stage. The findings also indicate that a slight temperature rise markedly increases the number of surviving Pm/bw^{V57e} flies.

From the cross, w^{a} fa^{no} spl rb/fa^{no} spl; SM1, Cy/Pm x y w^{a} N^{40}/Y; SM1, Cy, Dp(1;2)w^{51b7} bw^{V57e}/+, three classes of male offspring are expected if one disregards the X chromosomal genes. These three phenotypic categories may be expressed simply as: Cy/+, Pm/+, and Pm/bw^{V57e}. If one assumes equal viabilities, each class should comprise one-third of the total male progeny. The expected numbers and observed results are presented in the following table:

	Combined Cy/+ and Pm/+		Pm/bw^{V57e}		
Temperature	obs.	exp.	obs.	exp.	obs./exp.
26 ± 1°C	5,219	4,028	823	2,014	0.41
23.5 ± 0.5°C	10,888	7,788	794	3,894	0.20

The Pm/bw^{V57e} genotype, while considerably below the other two classes in viability, survives too frequently to warrant the lethal designation. A more appropriate description would be semi-lethal. It is possible that the higher than expected rate of survival of this particular heterozygote is due in part to a reduction in the number of its competitors by factors associated with the cross. All female zygotes (except rare cases of nondisjunction) are N^{40}/fa^{no}, a lethal combination permitting less than 0.01 imagoes. The Dp (1;2) w^{51b7}, which had been placed in the SM1, Cy, bw^{V57e} chromosome by Dr. W. J. Welshons, also carries N^{+}. Since the presence of N^{+} cancels the N^{40}/fa^{no} lethal interaction, there emerges a single group of female offspring namely, Pm/bw^{V57e}. Male progeny of this same genotype must compete only with other males and genetically similar females. The effect that additional classes and larger numbers of females will have on the relative viability of Pm/bw^{V57e} males is currently under investigation.

The following unpublished observations by Dr. W. J. Welshons on the phenotype of Pm/bw^{V57e} have been confirmed:

(1) Bodily dimensions range from clearly smaller than normal to approximately normal in size. The larger individuals are often characterized by a chubby (or bloated) appearance.

(2) Wings either fail to expland completely or, if unfolded, they tend to diverge and curl to an extent greater than that of Curly alone.

(3) Occasional patches of unpigmented microchaetae are another feature of the syndrome.

A limited number of tests show that these flies can be fertile.

The emergence of twice as many Pm/bw^{V57e} adults at the higher of the two temperatures employed provides one more example of the strong influence of environmental factors on the viability of a given genotype. It is interesting to note that Gowen and Gay (1933) found that the extent of variegation is diminished by increased temperature. From the results reported here it appears that the degree of semi-lethality of this particular brown-Variegated heterozygote manifests a like tendency.
(This work was done at the Biology Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee, operated by Union Carbide Corporation for the United States Atomic Energy Commission.)

Hunter, Alice S. and Sara Newball. Drosophila of old Providence Island.

Old Providence Island is in the Caribbean Sea at a latitude of 13°19' to 13°24' North and a longitude of 81°21' to 81°23' West. It is about 150 miles from the coast of Nicaragua and 350 miles from Cartagena on the coast of Colombia. The temperature

varies from 18-30°C. depending on the sun and wind, but averages 24°C. The terrain varies from the swamps and bays to wooded hills and mountains up to 1,152 feet above sea level. The wind is generally northeast from December to February, and the weather is calm from then until September. During October and November there may be hurricanes with strong winds mostly from the North.

The island is about 15 square miles, and there are about 3,000 human inhabitants. There are many fruits which might be natural breeding sites of Drosophila, such as the semi-cultivated mangos, papayas, coconuts, bananas, and the wild grapes, pineapples and guavas. However, despite the abundance of fruits, a total of only six different species of Drosophila were encountered in our collections made during the months of January and February.

Collections were made by sweeping over a bait composed of cut oranges, bananas and squash pulp. Whenever possible bait was placed in shaded protected areas, but in some places there were no trees, and it was impossible to avoid the wind. In fifteen sites the various species encountered in at least one of the sweepings were sorted and counted. The totals of the six different species found in the counted collections are as follows: D. melanogaster-simulans- 1,140, D. ananassae- 1,017, D. hydei (?)- 922, D. latisfaciaeformis- 493, and D. willistoni- 236. No attempt was made to determine the relative proportions of melanogaster and simulans. The identity of hydei is being checked in test-crosses with a University of Texas stock. Because of the lack of electricity on the island, all of the willistoni group flies collected were taken to Bogotá for identification. A sample of 50 males was checked by studying the genitalia. In addition 50 females were isolated in separate culture jars and the offspring were checked. In all the 100 cases the male genitalia were those of D. willistoni.

Comparing one collection site with another, considerable variation in the relative frequencies of the different species was encountered. For example:

	Free Town	Bush-pen	Camp
D. melanogaster-simulans	38	38	131
D. ananassae	349	20	68
D. hydei	54	1	250
D. latifasciaeformis	1	1	163
D. willistoni	0	32	0

The lack of variety of species is notable, but may be related to the isolation of this island and the winds.

(Biology Department, University of the Andes, Bogotá, Colombia)

Imaizumi, T. XXY strain derived from the wild Miyazu stock of D. melanogaster and its lethality.

Of the lethal strains in the preceding report (DIS 33 : 140), it is ascertained genetically that a strain derived from an X-rayed male is XXY. Perhaps primary nondisjunction occurred in a sperm of the X-rayed Miyazu male. The crossing tests are as follows:

Crosses	♀				♂				Totals
(1) Y/+/Basc ♀ x Basc ♂	B/+ 235		B 172		B 318			+ 0	725
(2) Y/+/Basc ♀ x w m ♂	B/+ red eye 212	orange eye 156		+ 157	B 134	w m 191		+ 0	850
(3) Y/+/w m ♀ x w m ♂	+ 572	w m 244	w 15	m 9	w m 523	w 0	m 8	+ 0	1371
(4) Y/+/y w m f ♀ x y w m f ♂	+ 449	y w m f 231	y w 1	m f 32	y w m f 413	m f 9		+ 0	1150
	m 7	(rb)* 8							

*A new character found in cross (4); it represents reduced bristles, not forked, phenotypically and its chromosomal constitution is XXY.

It is noticed firstly from the crosses (3) and (4) that the crossing-over between two X-chromosomes is very suppressed in this XXY strain. Next, at meiosis, three kinds of segregation are expected in the XXY females: (a), (Miyazu-X, introduced-X) and (Y); (b), (Miyazu-X, Y) and (introduced-X); and (c), (introduced-X, Y) and (Miyazu-X). But we can prove that the last segregation (c) never occur in this strain by the tests in F_1 flies of the cross (2). In F_1 of this cross the chromosomal constitution of the females with hetero Bar (orange eye) phenotype is all XX and that of the females with wild phenotype all XXY. Further, it is clear that the wild male never appears in all cases. The XY zygotes (wild genotype) are not formed from the segregation (c) and the XYY males (wild genotype) derived from the segregation (b) would be lethal in this strain, though one knows that the XYY males are viable in general.

The percent mortality in F_1 of several crosses is shown in the following table. The lethality of the XXY strain is given in the top cross (1) and that of the attached-X strain in the next cross (2); and the other three are controls. Percentages per the first total eggs tested are indicated in each stage.

Crosses	Total eggs tested	Mortality in egg					Mortality in larva	Mortality in pupa	Total mortality rate
		Total	Items of stages* U	C-B	Bk	T			
(1) Y/+/Basc♀ X (XXY) Oregon-RS	757	66.4	1.3	19.5	29.2	16.4	15.0	1.7	83.1
(2) g^2 ty/y (Attached-X)♀ X Oregon-RS	745	37.9	2.8	27.1	4.6	3.4	18.4	12.9	69.2
(3) Basc/Basc♀ X (XX) Oregon-RS	727	10.3	4.7	0.4	1.0	4.3	15.5	0.3	26.1
(4) Oregon-RS♀ X Oregon-RS	542	1.3	0.9	0	0.4	0	19.9	0.4	21.6
(5) Miyazu♀ X Miyazu	673	3.3	2.5	0	0.2	0.6	11.3	0.9	15.5

*U; unfertilized eggs; C-B, Bk and T indicate primary, middle and late stage in the embryonic development respectively.

Regarding triple-X females, it is revealed that they mostly die in the pupal stage, but some in the late larvae as indicated in the progeny of attached-X strain. But in this XXY strain the mortality rate in the pupal stage is evidently low as compared to the case of the attached-X. On the contrary the mortality in middle or late embryonic stages is higher. This fact suggests that the time of death of the triple-X females in this XXY strain is not in pupal stage, but it would be in embryonic stage, perhaps in the middle of the stage. Finally, the YY individuals die in the primary stage of embryonic development both in XXY and attached-X strains.

Ives, P. T. More data from ras^2/ras^4 and y/y^2 recombination tests.

In 1951 I reported a count of 66,907 flies from ras^2/ras^4 which showed no ras^+. In 1961 I have added 69,385 flies to that total with the same result. No evidence of pseudo-allelism or conversion has appeared in these 136,292 flies. Both series of tests were done with outside markers, with free X-chromosomes and without autosomal Inversions. During the summer of 1961 Dr. Hexter and I plan to test ras/ras^4 with outside markers in free X's and with Cy and Ubx^{130} present. The 69,385 flies recorded here also tested y/y^2. No y^+ flies appeared.

Jacobs, M. E. Influence of ebony and + alleles on oxygen consumption and egg production in D. melanogaster. Studies of flies homozygous and heterozygous for the ebony gene found in a wild population, with randomization of other gene loci, showed heterozygosity to increase significantly the rate of pupal oxygen consumption and adult egg production.

Kato, M. Influences of essential fatty acids on the growth and egg productivity of D. melanogaster. Influences were tested by addition of the so-called essential fatty acids or vitamin F to the culture medium which was prepared by extracting lipids from the homogenized yeast with absolute alcohol and ether. In this medium the emergence rate of the adult fly was 8.0±4.0% on an average while in the standard medium it was 86.33±1.57%. When whole EFA were added to the medium it increased to 44.16±0.93%. When only EFA with double bonds at 6,7- and 9,10- position were added, the rate approached the level obtained with whole EFA, namely, 36±0.55%. In the EFA-deficient medium, most of the larvae died before pupation and most of those that pupated did not survive to adulthood. Neither abnormality in morphology nor that in sex-ratio were found. Daily ovulation of females emerged in the EFA-deficient medium is very low, being 9.34+1.13% as compared to that of 28.55±2.09 produced by the female reared in the EFA added medium.

Khishin, Aziz F. Induction of mutations in D. melanogaster by "immersion" in solutions. The experiments designed to test the possibility of inducing lethal mutations in Drosophila melanogaster by "immersing" larvae or pupae in formaldehyde solutions gave definite positive results (Khishin, published and unpublished). However, it should be questioned whether in these cases the formalin itself or even a derived product is the sole mutagenic agent, or whether the mere immersing has an effect and thus should be held responsible for all or some of the observed changes.

To test the second possibility a set of experiments were started in which tap water, saline solution prepared with distilled water, and formaldehyde solutions were used as media for immersion. The same procedure was followed for the three agents used, and larvae were treated for 30 minutes and 60 minutes. In all cases dominant lethality (D.L.) was calculated over certain period. Preliminary results indicate the following:

1. Tap water induces more D.L. than either saline (0.05% Na Cl) or 10% formaldehyde solutions.
2. Saline solution induces about the same percentage of D.L. as 10% formaldehyde solution when either is used for 30 minutes.
3. Saline induces more D.L. than formaldehyde when either is tested for 60 minutes.
4. 10% Formaldehyde used for 30 minutes gives about 2-3 times as much D.L. as when applied for 60 minutes.
5. Saline solution used for 30 or 60 minutes gives about the same result.

Mead, Charles G.*, and Allen S. Fox. The characterization of the deoxyribonucleic acids of Drosophila melanogaster. DNA was isolated from lyophilized Oregon R flies by a modification of the Kay, Simmons and Dounce procedure (1952). Approximately 2 mg. of DNA were recovered per gram dry weight of flies. The isolated product had an E(P) of 6930, exhibited a 20% increase in O.D. at 260 mμ upon alkaline denaturation, and was unusually low in viscosity. Upon precipitation of the isolated product with cold ethanol, two types of DNA were observed. One of these was fibrous, typical of most DNA's, and the other was of a flocculent nature. After exhaustive deproteinization the two types of DNA retained their differences.

Perchloric acid and formic acid hydrolysates of the isolated DNA, when subjected to paper chromatography, exhibited an exceptional UV-absorbing spot. This unusual compound was identified as 5-methylcytosine by chromatographic and spectrophotometric means. The two types of ethanol precipitable DNA's, one fibrous and the other flocculent, were analyzed for their 5-methylcytosine contents. A preparation of DNA was precipitated with cold ethanol and the fibrous DNA collected by winding the fibers on a glass rod. The fraction which could not be collected in this manner was considered flocculent DNA. Each fraction was hydrolyzed with perchloric acid, the hydrolysates chromatographed on paper, the UV-absorbing spots eluted, and the molar concentration

of 5-methylcytosine and thymine measured spectrophotometrically. The molar ratio (5MC/T) of the fibrous fraction was 0.165±0.008 (n=4), and that of the flocculent fraction was 0.234± 0.007 (n=6). Thus, the pyrmidine composition of these two fractions is definitely different.

Ion exchange chromatography of a phosphodiesterase digest of whole DNA yielded five UV-absorbing peaks. The molar concentrations of these peaks, as calculated from their respective molecular extinction coefficients at 260 mμ, are as follows:

Peak No.	Nucleotide	μM	
1	Deoxy-5-methylcytidilic	0.79	2.42
2	Deoxycytidilic	1.63	
3	Deoxythymidilic	2.78	
4	Deoxyadenylic	2.76	
5	Deoxyguanylic	2.47	

The existence of 5-methylcytosine in the DNA of Drosophila makes possible a variety of experiments which might lead toward the elucidation of the relationship between the genetic units and the chemistry of DNA. We now have a non-randomly distributed label in a DNA which can be defined genetically with great accuracy and maniuplated with ease. An attempt to identify this label with specific genetic units could yield valuable information.

(Supported by grant C-2440 from the National Institutes of Health, U.S. Public Health Service. *Predoctoral Fellow, National Institutes of Health, U.S. Public Health Service).

Meyer, Helen U. and Michael L. Criswell. Crossover analysis of sex-linked mutations induced in oogonial cells by repeated treatments with 4000r of X-rays.

When heavy doses of X-rays are divided into instalments and given at 4-day intervals, only the potential chromosomal breaks from the same irradiation can collaborate with one another. The proportion of chromosomal rearrangements is therefore determined independently by each of these particular instalments. This method of fractionated treatment lowers the mortality of the treated cells considerably in comparison with that with that occurring when the same total dose is given as a continuous, "acute" treatment. Such a method has been used in many of our experiments in which mutations induced in gonial cells were studied, where a rather heavy X-ray dose is needed to counteract the relatively low X-ray sensitivity of these cell stages. Not many gross changes in chromosomal structure were expected when irradiating in this manner; however it was desirable to get more information on this question. From an experiment in which a total dose of 24,000r, given in 6 instalments, resulted in 15.2% sex-linked lethals, we selected nine different cases of sex-linked mutations at random, derived from eggs laid 8-12 days after the last irradiation, for analysis of the crossover pattern. The mutation-carrying X-chromosomes were originally of isogenic origin, had the normal gene sequence and were marked by sn oc. One was a visible mutation, one a detrimental (about 15% of expected survivors), four near-lethals (less than 10% of expected survivors), and three fully lethal. In only one out of these nine cases had some rearrangement of the gene sequence occurred; this had resulted in a small inversion which was connected with a lethal. In the remaining 8 cases the crossover values were found to be in reasonable agreement with the expected map frequencies. Similar, more recent studies on mutations recovered from gonial cells by only one treatment with 4000r seem to agree with these findings that intrachromosomal rearrangements may be recovered from gonial cells, but in a much lower proportion than when a similar dose is given to mature spermatozoa.

(This work was supported by a grant to H. J. Muller and associates from the Public Health Service, Contract RG-5286 (3), and a grant from the National Science Foundation Summer Programs for Secondary School Students.)

Meyer, Helen U. and Evelyn R. Meyer. Sperm utilization from successive copulations in females of Drosophila melanogaster.

To learn how sperm from successive copulations is utilized, young virgin females homozygous for the second chromosome markers cn bw sp were mated first with

homozygous cn sp males and then remated to homozygous bw sp males. Only one copulation with each of these two types of males was allowed, and the females isolated immediately afterwards.

An interval of 4-5 days was unfortunately necessary between the two inseminations, since the females refused to mate for a second time before a sizable number of fertilized eggs had been deposited. It was found that the length of this interval varied with temperature and with the type of culture medium used. That various strains behave differently in this respect has been pointed out by Ehrlich in a similar study (D.I.S. 33:129-130, 1959).

39 females, which had been observed to have copulated once with each type of male and had been immediately separated from them, were bred individually and transferred to fresh culture vials every 24 hours for 12 successive days. After this time only one of them still laid fertilized eggs. All offspring from these daily broods were classified for phenotype, cn sp progeny indicating fertilization by the first male, and bw sp by sperm from the second one.

Two parent-females gave only one type of offspring, cn sp in one instance and bw sp in the other one, even though they had been observed to have copulated with both types of males. This could be explained by either non-functional sperm (as in XO males) or by copulation without insemination.

The results from the remaining 37 doubly inseminated females are summarized in the following table:

EGGLAYING PERIOD	AV. NO. FERTILE P-♀♀	AV. NO. F_1 PER P-♀♀ PER DAY	PHENOTYPE OF F_1		% F_1, FIRST INSEMIN.
			cn sp	bw sp	
BEFORE 2nd Insemination (4-5 days)	31*	19.5	2936	-	100 %
AFTER 2nd Insemination					
days 1-2	36.5	35.4	64	2521	2.48
3-4	33.0	24.3	27	1578	1.68
5-6	27.0	13.6	13	719	1.78
7-8	19.5	5.1	4	196	2.00
9-10	11	3.5	1	76	1.30
11-12	6	3.8	1	45	2.17
Totals, days 1-12			110	5135	2.1

*No record kept on the no. of F_1 from 6 other P-♀♀ during this period, which are included in the second part of this table.

Thus an average of only 2.1% of the total offspring obtained after the second mating was derived from sperm retained from the first insemination. However, this frequency did not vary significantly from brood to brood except for a slight drop after the first two days. These findings therefore do not support the view that spermatozoa are stored in the order in which they are received, but imply that they are mixed in the storage organs of the females and used at random to fertilize the eggs, in agreement with similar results obtained by Ehrlich as described in the report quoted above.

At the end of the observation period only 6 of the initial 37 females were still reproducing. Since the females were not yet very old, this was undoubtedly due to sperm exhaustion.

Fewer offspring resulted from the first insemination than from the second one. This might have been due to a variety of factors: the lower egg production of very young females, possibly a not yet fully developed storage capacity for sperm, or discharge of stored sperm before or during the second copulation. It also might have been due to the different genotype of the first male.

On the basis of our data it is possible to make a tentative estimate of the number of eggs which can be successfully fertilized by the amount of sperm deposited during one copulation. By adjusting the number of progeny from the first male by addition of an estimated 568 (for those 6 females for which no record was kept during the first period), a total of 3614 offspring were obtained from the first male, and 5135 from the second male. Dividing their average by 37, we obtain an estimate of

118 offspring from one combination; or, if one considers the result of the second mating to be more representative for the middle period of reproductive life, the number would be 140 successfully fertilized eggs.
(This work has been supported by a grant to Dr. H. J. Muller and associates from the U.S. Public Health Service, Contract RG-5286(3)).

Novitski, E. Post-treatment of irradiated sperm by low temperature. In a talk given before the Conference on Problems of General and Cellular Physiology in 1949, I made the statement that post-treatment of irradiated sperm by low temperature caused an increased recovery of sex-linked lethals. The transcript of this talk was subsequently published (American Naturalist, LXXXIII, 185-193) without the inclusion of any supporting data. Since that time I have been asked several times about these experiments.

In these runs, Basc ♀♀, previously inseminated by Canton-S ♂♂, were irradiated in order to remove the ambiguity of possible differential sensitivity of the stages of spermatogenesis. A control series, irradiated with 3600r at 25°C, gave 128/1246 (9.7%) lethals; a parallel series, held at 0.5°C during the irradiation, gave 34/138 or 24.6% lethals.

In the more extensive sets described below the dose was decreased to 1800r. The controls at 25°C gave 54/1166 (4.6%) lethals. An unirradiated control exposed to -1°C for 14 hrs. produced no lethals in a total of 877 tests. When the cold treatment (6 hrs.) preceded the irradiation (but with an hour and a half separating the two) there was no appreciable change in the lethal frequency (50/1172 = 4.3%). In two runs the females were kept at 0°C during the treatment and the lethal rate jumped appreciably (78/1113 = 7.0%; 99/1121 = 8.8%). Finally, in a run in which the females were exposed to the low temperature immediately after X-raying, the lethal rate was 11.7% (11/94). Although statistically significant, the low numbers in this last case, which was the only one involving bona fide post-treatment only, suggested repetition. Unfortunately, at this stage, desemination (which was undoubtedly responsible for the low numbers in this last experiment) became a serious problem and attempts at repetition failed dismally. This line of experimentation was then abandoned, although the effect of cold temperature in deseminating females was duly investigated and published.

Parker, D. An apparent incompatibility among seemingly normal members of the species D. simulans. Peculiar results were observed in experiments with D. simulans whenever a cross involved the mutant vermilion. Difficulties with D. simulans crosses cropped up continuously when vermilion was mated with the mutants yellow-white, black, sepia, scarlet peach, peach-hairless, plum, and with various wild type stocks. These difficulties were due to rather unpredictable abnormalities in the development of the offspring.

The crosses using vermilion males were moderately successful with the only deviation from normal being in the reduced amount of progeny from each cross. But the results when using vermilion females were more noteworthy. In roughly 70% of all the vermilion female crosses, development went no further than the egg stage. In approximately 15% of all crosses involving vermilion females, the development progressed until death occurred in the larva stage. The time of death established no predictable time pattern. Death occurred any time between the first instar and pupation. In the remaining 15% of the crosses, adult offspring emerged only to die sometime within the first five days. Even when such adult progeny did appear, they were much reduced in number from what is normally found.

Roberts, Paul A. Bristle differentiation in genetic mosaics of D. melanogaster. It was demonstrated by Stern (Proc. 9th Intern. Congr. Genetics. Part I:355, 1954), using gynandromorphs, that achaete (ac) tissue is autonomous in mosaics. That is, in flies in which the site of a posterior dorsocentral bristle is ac tissue, a bristle will usually not differentiate even when most of the surrounding tissue is ac^+. Conversely, when the site of the bristle is ac^+, differentiation is always initiated regardless of the amount and distribution of surrounding ac tissue.

Out of 1600 duplication carrying male offspring of the cross of females, Dp(w^{vc})6094b/y w f:=/Y, by males, y ac w^a $ct^6f \cdot Y^S$ /Y^L, 110 had some loss of the duplication (carrying non-yellow, y^+, and non achaete, ac^+) and were mosaics involving the dorsocentrals. In this genotype, 99% of the male progeny not carrying the duplication (patroclinous males) had both anterior and posterior dorsocentrals missing, so both of these sites were scored. Of these 110 mosaics, 91 exhibited autonomy as described above, and 19 exhibited non-autonomy in which dorsocentral bristles differentiated in sites in ac tissue close to ac^+ tissue. Stern interpreted this as due to spread of ac^+ material into the ac tissue patch. However, 12 of the 91 autonomous cases had similar proximity of ac^+ tissue to the bristle site with maintenance of ac autonomy. Seven cases were observed in which a dorsocentral bristle differentiated at an abnormal site near a bristle site occupied by achaete tissue, but apparently within a potential area of differentiation.

Results are consistent with Stern's observations and his interpretation that ac and ac^+ are not establishing a regional singularity but responding to a "prepattern" present in both genotypes.

Sandler, L. and C. W. Cotterman. A possible interpretation of the conversion of X chromosome by SD.

In the presence of SD (which is located on chromosome II and conditions abnormal segregation of this chromosome pair in males), some X chromosomes may be specifically converted into suppressors of SD action (Sandler and Hiraizumi, 1961).

It is not known by what mechanisms either (1) the conversion of the X chromosome takes place, or (2) the converted X chromosome suppresses SD action. One possible supposition that can account for both of these effects is as follows. It may be imagined that the conversion results from the X chromosome physically acquiring a part of the SD locus. This modified X chromosome, in subsequent generations, can pair with the SD region of chromosome II in SD heterozygotes and thus prevent SD from pairing properly with SD^+. This should indeed suppress the phenomenon of segregation-distortion because synapsis of SD and SD^+ is known to be necessary for distortion (Sandler, Hiraizumi and Sandler, 1959).

The only test of this notion that immediately suggests itself is to see whether there are any differences in the segregation of the X and chromosome II in SD heterozygotes, according to whether or not the X chromosome has been converted.

Accordingly, males heterozygous for SD and In(2LR)Cy (Cy, itself, suppresses SD action by failing to pair properly with SD and is used here so as to maximize the probability of X-II synapsis) wither either a modified X or an unmodified X, were crossed to cn bw females. The results were as follows:

	Progeny			
Type of X	Cy♂♂	Cy♀♀	Cy^+♂♂	Cy^+♀♀
Modified	229	259	265	246
	(.23)	(.26)	(.27)	(.25)
Normal	315	288	315	277
	(.26)	(.24)	(.26)	(.23)

It is clear that X-II segregation is the same irrespective of whether or not the X has been modified. Thus we must suppose that either the suggested hypothesis is incorrect or that pairing between the X and SD, while sufficient to suppress SD action, is not of such a kind as to affect the pattern of segregation.

Sederoff, R. and E. A. Carlson. The relation between allelic phenotype and allelic localization within the dumpy region.

A specific sublocus has been established in the dumpy gene for the ov mutants. These ov mutants exhibit two phenotypic effects of the dumpy gene, the oblique wing, written as o, and the bristle disturbances and protuberances of the thorax called vortex, written as v.

A series of six independently arising ov mutants (ov^1, ov^n, ov^x, ov^{51f}, ov^{52b}, ov^h) were localized within the genetic map of the dumpy region. A modified "four-point" test using the outside markers -- echinoid (ed) at 11.0 and clot (cl) at 16.5 was used in the mapping procedure. The position of these alleles was determined with respect to two other alleles of the dumpy region, the thoraxate (lv) allele on

on the left of ov^1 and the vortex (v) allele on its right. In all instances the other ov alleles were mapped between 1v and v. These localizations establish a sublocus within the dumpy gene which appears to be specific for the ov expression. No other alleles have been localized at this site. Therefore, the region may be referred to as the ov sublocus. (See Table 1 for the summarized results.)

One of the ov mutants, called ov^h (dumpy-humpy-like) shows a more extreme effect than the other ov alleles, and it is a facultative lethal in the homozygote. It is not lethal, however, in the heterozygous compound with any of the alleles of the dumpy region containing the lethal factor (e.g. l, ol, lv, olv). The localization of this allele between thoraxate, lv, and vortex, v, suggests that the ov^h mutant might be a minute deletion of the ov sublocus, or that it might occupy a separable site within the region.

These localizations indicate that mutation at the dumpy locus will usually be specific in expression for that portion of the map which is affected. A possible exception to this may be found for the olv alleles. These mutants are characterized by a loss of the total function of the dumpy gene. In phenotype they resemble deficiencies for the dumpy region. These extreme mutants could possibly be located anywhere on the pseudoallelic map. Localization of a series of olv alleles is now in progress (Southin and Carlson, unpublished).

The localization of the ov alleles to a specific sublocus makes possible an investigation of the fine structure of this portion of the dumpy region. A selective technique has been designed (see p.___, this issue) which will be used to investigate this possibility of fine structure. Southin (1961, unpublished) has obtained recombination between two similar oblique alleles, o^2 and o^{bm}. Resolving power of this test was approximately 1×10^{-5} and we anticipate possible resolution with the selective technique to reach 1×10^{-7}.

Table 1
Localization of the ov Alleles

Trans Alleles	Verified "Conversions"	Verified Single C.O.'s	Total Count
lv^1/ov^1	1	6	66,009
lv^1/ov^h	0	1	3,548
lv^1/ov^x	0	2	22,668
lv^1/ov^n	1	2	22,766
lv^1/ov^{51f}	0	1	34,544
lv^1/ov^{52b}	0	1	21,477
v^2/ov^1	0	7	35,200
v^2/ov^h	0	1	2,955
v^2/ov^x	0	2	7,894
v^2/ov^{52b}	0	1	20,820
v^2/ov^{51f}	0	1	7,298
v^2/ov^n	0	2	4,539
TOTALS	2	27	249,718

This work is supported by Grant G 14222 from the National Science Foundation.

Seto, F. The relative constancy of phase specific action of recessive lethal factors in D. melanogaster.

In a recent paper Hadorn (1959 Arch. Jul. Klaus-Stiftg., 34:234-239) reported that the phase specificity of 19 recessive lethals had remained unchanged over the past 7-8 years. Similar observations made in this laboratory tend to confirm Hadorn's general conclusions. Several strains of second chromosome recessive lethals, which manifest developmental effects in the larval-pupal and pupal stages, have been maintained in a balanced condition (^{Cy}/le) for several years. During this interval the period of action of various lethals had been determined on several occasions in the course of various experiments, either by counts or by direct observations of their visible effects. In most of the lethals the phase specific action and characteristic phenotypes remained unchanged but a few had lost their larval-pupal or pupal effects and were later discarded. A summary of the observations on the various lethals are given below:

Period of action	Lethals	1951/52	1953/54	1956	1958	1960
Larval-pupal	N-59,N-1,X-11	*	--- D			
	X-3	*	*	*	---	(*)
	N-61,N-51	*	*	*	*	*
Early pupal	N-50	*	*	*	---	D
Pupal	N-32	*	*	*	*	*
	Co-7,Co-3A		*	*	*	*
Late pupal	N-42(N42A)	*	(*)	(*)	---	*
	N-13			*	---	---
	N-1A		*	*	*	*
Late pupal, adult	N-45		*	*	*	*
	N-55			*	*	*

* phenotype typical (*) phenotype altered --- phenotype lost D culture discarded

Lethal X-3 lost its L/P effect by 1958 but by outcrossing and re-isolating new lines it regained its L/P phase specificity but with an altered phenotype. N-42 initially manifested a late pupal effect which was lost or replaced soon after by a larval-pupal phenotype. By 1958 the L/P effect weakened and was lost but one of the outcrossed lines had regained the original pupal effect which it still maintains.

In another study 37 second chromosome recessive lethals obtained from wild populations were tested for their period of action initially toward the end of 1959 and a second time at the beginning of 1961. The ontogenetic distributions of lethality of the 37 lethals in the two counts were:

year	(phase of action)	E	E/L	L	L/P	P	Total
1959		1	5	22	5	4	37
1961		4	5	20	6	2	37

A X^2 test of heterogeneity indicates that there is no significant difference in the distributions. A comparison of the individual lethals showed that 13 of the 37 lethals displayed a change in the stage of lethal action. However, only two of the thirteen showed marked changes (from the P or L/P to the E stage) whereas the others showed only minor shifts to an adjacent stage. The results of the observations on these 11 lethals are summarized below:

Shift to an earlier stage		Shift to a later stage	
E/L to E	B-55	E/L to L	B-6
L to E/L	B-19,B-20,B-39	L to L/P	B-14,B-27
L/P to L	B-33	E/L/P to P	B-23
P to L	B-58		
P to L/P	B-52		
	(7)		(4)

24 lethals did not show any change in phase specificity. Of the changes observed, most of them were minor changes are are not significant. It is known from earlier studies that cultural conditions and differences in genetic background can alter the time of expression but in general the phase specificity tends to be relatively constant.

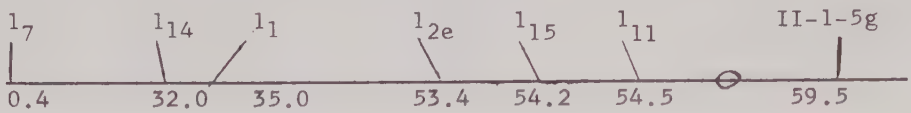
(Part of this work supported by NSF research grant G106-99)

Spiess, E. B. and Helling, R. B.
Linkage of chromosome II lethals in D. melanogaster.

A series of lethals on chromosome II were extracted from a population cage of D. melanogaster descended from the control #3 population of Bruce Wallace, which was initiated with Oregon-R in 1949 and had been maintained for at least 170 generations at 25°C in a plastic population box before this laboratory obtained subsamples from it. Lethals were extracted in conjunction with an attempt to obtain "good viability" chromosomes (done by Mr. Archie Allen using the Cy L/Pm technique), and they were maintained in balanced condition with Cy L. Salivary analysis revealed no aberrations. The frequency of lethal chromosomes in the cage was estimated at 0.175, but linkage tests showed that the lethals were all in the left arm of chromosome II. Mapping of lethals was done in two sets of crosses: the first set utilizing markers al pr c sp and the second set using markers in either right or left arms depending on where the

first set of crosses placed the lethal: al dp b pr and pr c px sp. Females heterozygous for lethal/recessive markers were backcrossed to males with these markers homozygous plus a dominant such as Cy or Bl. All crossover class♂♂ containing the dominant were backcrossed to the lethal/Cy L. By counting 100 flies in the progeny and finding no wild type, lethality was classified.

The following linkage map was obtained from classifying ten lethal chromosomes; a single lethal obtained as a spontaneous mutant in another experiment (Allen) (II-1-5g) was placed in the right arm:



By testing lethals for allelism, l_7 occurred four times, twice with another lethal locus (for example l_2 was lethal at the l_7 locus plus l_{2e} at 53.4); l_1 occurred twice; and in addition two other multilocus lethals were obtained which have not yet been located exactly. With such high frequency of certain lethals and high frequency of multilocus lethals, it might be inferred that high fitness must be conferred upon lethal heterozygotes in this population. Relative viability tests are now being made by the senior author. Certainly the non-random distribution of these lethals would imply possibly blocks of genes in the left arm of chromosome II which might be heterotic.

Lethals obtained by similar tests on chromosome III are currently being carried out also. Allelism is very high but linkage analysis has not been completed.

Stern, C. and E. Sherwood. A search for maternally influenced sex-ratio in Drosophila melanogaster.

In order to find out whether females of Drosophila melanogaster or Drosophila simulans caught in nature would produce offspring of unusual sex-ratios, traps were set out at various localities, far enough away from experimental laboratories to exclude the possibility of trapping laboratory-bred flies. Each female was put singly into a culture bottle and allowed to lay eggs for seven days.

Of a total 626 females 606 produced offspring in a 1♀ :1♂ ratio, 14 females gave an F_1 in the ratio of 3♀:1♂, and 6 females had only female progeny. Each one of the all-female progeny was first mated to D. melanogaster, then to D. simulans males, and none were fertile. One of the mothers was mated to D. melanogaster males and gave offspring in the normal sex-ratio.

The results fit well with Sturtevant's findings of 1929, where he reports 10-40% all female hybrid offspring from D. melanogaster ♀ x D. simulans ♂♂ in crosses done in the laboratory. In the reciprocal crosses, i.e. D. melanogaster ♂♂ x D. simulans ♀♀, he obtained 2% all-male hybrids. None was found in our experiments.

Whether the hybridizations occurred in nature or within the trapping bottles is unknown. In any case, no maternally determined "sex-ratio" condition was found in the sample of 606 tested females.

Strangio, V. A. Radiosensitivity to certain breakage aberrations during spermatogenesis in D. melanogaster.

A re-investigation of radiosensitivity during the spermatogenesis of D. melanogaster has been made utilising a doubly-marked Y-chromosome, y^+YB^S (see Brosseau et al, 1961. Genetics 46:339-346 for full description). Recently emerged Canton-S males carrying this Y-chromosome were irradiated with 1000r and then mated daily for twelve days afterwards either to three y apr ec females (Series I) or to four attached-X y v f car females (Series II). In Series I, the regular offspring were wild type females and apr ec - Bar males; in Series II, v f car - Bar females and wild type males. The improved technique described here allows the following irradiation-induced aberrations to be detected simultaneously:

(a) sex-chromosome losses as:
XO males in Series I, phenotypically "y apr ec".
XXO females in Series II, phenotypically "y v f car".

(b) individual Y-chromosome marker deletions as:
XY^- males in Series I, where loss of y^+ yields y apr ec - Bar ("yellow") males or loss of B^S results in apr ec ("non-Bar") males.
XXY^- females in Series II, loss of y^+ produces y v f car - Bar ("yellow")

Day	1	2	3	4	5	6	7	8	9	10	11	12
SERIES I												
Total Offspring	2390	2207	2637	2125	1625	845	420	409	1862	1858	1481	1747
% "y apr ec" ♂♂	0.04	0.09	0.15	0.56	0.98	1.07	1.43	-	0.21	-	-	-
% "yellow" ♂♂	-	0.05	-	0.09	0.06	0.83	0.95	0.24	0.05	0.16	-	-
% "non Bar" ♂♂	0.04	0.09	0.04	0.19	0.25	0.71	0.48	1.71	0.05	0.11	0.07	0.06
% "Bar" ♀♀	0.04	-	-	-	-	0.24	0.95	0.24	-	-	0.14	-
SERIES II												
Total Offspring	1278	2235	2330	2287	1245	952	380	194	997	1709	1582	1685
% "y v f car" ♀♀	0.31	0.22	0.26	0.48	1.37	1.05	2.63	1.55	0.10	0.12	-	-
% "yellow" ♀♀	-	-	-	-	0.16	0.32	2.63	1.03	0.20	0.23	0.13	-
% "non Bar" ♀♀	-	0.04	0.26	0.57	0.64	1.47	3.42	4.12	0.20	0.12	-	-
% "non yellow" ♀♀	-	-	0.04	0.17	0.08	0.11	0.79	0.52	-	-	-	-
% "Bar" ♂♂	-	-	-	-	-	0.11	0.26	-	-		-	-

females or loss of B^S yields v f car ("non-Bar") females.

(c) non-disjunction of X and Y as:
XXY females in Series I, phenotypically "Bar".
XYY males in Series II, also "Bar".

(d) large interstitial X-deletions as:
XXX⁻ hyperploid females in Series II, phenotypically "non-yellow" and usually also showing one or two of the v f car markers.

The results are summarized in the accompanying table and are similar to those published as separate experiments by Ives (1960), Sävhagen (1960) and Chandley & Bateman (1960). The egg-laying period of the females is kept constant for each brood so that the relatively low adult numbers on the seventh and eight days are probably a reflection of maximal dominant lethal induction. This has been confirmed by direct egg mortality counts which incidentally also reveal a remarkable consistency in the radiosensitivity patterns for individual males. The aberration frequency versus brood curves for sex-chromosome loss, Y-deletions and X-deletions are all essentially similar and reach peak level on the seventh or eight day after irradiation. When these are compared with the onset of induced non-disjunction of X and Y chromosomes which must occur before their separation during the reductional meiotic division, it appears that the chromosomes exhibit their greatest radiosensitivity to the induction of at least these types of abberation during the early meiotic stages i.e. in the spermatocytes. Further investigations are in progress with ring and inverted X-chromosomes.

Tates, A. D., and F. H. Sobels.
The genetic effects of post-radiation treatment with cyanide in pupal spermatids.

In earlier experiments it was observed that treatment with cyanide following high-intensity X-irradiation (2200r/min.) resulted in a significant increase of the frequency of sex linked lethals and translocations in stages with greatest sensitivity to X-rays (Sobels 1960). These stages, presumably corresponding to spermatids, were sampled by means of the "brooding technique" after treatment of adult males. The extent to which cyanide enhanced the mutation rate showed, however, a considerable variation from experiment to experiment. This was felt as a serious handicap at a further analysis of the post-treatment effect. Assuming that susceptibility to the action of cyanide is restricted to one particular stage only, imperfections of the brooding technique in specific sampling could be responsible for the variation mentioned above. Since according to observations of Khishin (1955) and Oster (1955) radiated spermatids can be obtained in a more selective manner by treating 48-hour pupae, we investigated whether post-treatment of 48-hour pupae would yield more uniform results. Also, this method would be less time consuming than that of sampling spermatids by the brood technique from treated adults.

Male pupae of the genetic composition In(1)dl-49, y B/sc^8·Y; bw^D were irradiated with either 1200 or 2000r at a dose rate of 3000r/min., 100 KVP, 3.9 mA, without additional filtration. Ninety seconds after completing the radiation, part of the pupae were exposed to hydrocyanic acid, equivalent to 37.5 mg KCN, at a rate of flow of 100 ml/min. during 5 minutes. Preliminary tests had shown that with this procedure sterility was approximately 25% and mortality less than 10%. After hatching the males were mated individually to three females of the Y^SIn(1)EN·Y^L, y; st stock. Their progeny was tested for the incidence of sex-linked lethals and translocations of the II-III, Y-II, Y-III and Y-II-III types, according to Muller's (1954) multipurpose method. The results are presented in tables 1 and 2.

Table 1

Experiment	Dose (r)	Sex-linked lethals n. chrom.	leth.	% leth.
R_1	1200	968	129	13.3
R_1-CN		914	139	15.2
R_2	2000	436	95	12.8
R_2-CN		293	69	23.5

Table 2

Experiment	Dose (r)	Translocations (total number) n gametes	transl.	%transl.	Translocations II-III transl.	%transl.
R_1	1200	424	71	16.7	45	10.6
R_1-CN		483	96	19.9	54	11.1
R_2-	2000	260	57	21.9	32	12.3
R_2-CN		199	59	29.6	34	17.1

The data show that post-treatment enhanced the frequency of both lethals and translocations, though not in a significant manner. Compared to the observations on treated flies, the modification is less pronounced. The method of treating pupae does not offer, therefore, technical advantages for a quantitative study of the cyanide effect.

Terzaghi, Eric and E. Novitski. An attempt to produce fertile "transformed" males.

Because of its obvious technical utility and its intrinsic developmental genetic interest, a number of attempts have been made in the past, by several investigators, to obtain a fertile "transformed" male (X/X; tra/tra). The authors have performed a large-scale radiation experiment in an attempt to produce the desired "transformed" male.

In a large scale radiation experiment, it is of great benefit to have an experimental set-up in which a large number of individuals may be tested for the desired characteristic with a minimum of time expended. Towards this end, the following mating scheme was designed, in which the progeny of the last cross select themselves in respect to the desired characteristic, fertility.

$yf:=(RA)/Y^S$, y^+; +/tra X X^D+ $B^S \cdot Y^S/Y^L$, y^+; +/+

↓

irradiated with 4000r

$w^a v/Y$; tra/tra or +/tra X $yf:=(RA)/B^S \cdot Y^S$; +/tra or +/+

↓

$w^a v/B^S \cdot Y^S$; +/+ or +/tra or tra/tra, $yf:=(RA)/Y$; +/+ or +/tra or tra/tra

Among the progeny of the first mating, no fertile males are expected, hence, rigorous and frequent virgin collection is eliminated. Among the progeny of the second mating, no fertile males would normally be expected except in that case when the appropriate dominant mutation had been induced, or the appropriate recessive allele of "transformer" had been induced.

Contingencies which would produce undesired fertile males, such as breakdown of the double X (reversed acrocentric) or non-disjunction of the X and Y in the irradiated male, have been met, respectively, by making certain that both arms of the double X contain lethals and including the Bar Stone fragment in the chromosomal complement of the mates of the irradiated males. Thus, in the latter case, any zygote getting both the X and the Y from the male parent, would get either the Bar Stone fragment or the double X from the mother. The former combination produces a sterile male, and the latter is a super-female.

In order to attain the maximum yield of offspring per female and to conserve labor, a culture technique suggested by Spencer was employed. In this system, 100 to 200 females, plus the appropriate number of males, were placed in a quarter pint milk bottle with the standard cornmeal-molasses medium. They were allowed to remain for two days, and then were transferred to a fresh bottle. Two days later, a small wad of Kleenex (two sheets), saturated with a thick solution of fresh bakers yeast, was placed on the food in the bottom of the bottle. Females were kept and used until they died, only making certain that the initial number of flies per bottle was maintained, by combining bottles when necessary.

To date, approximately 150,000 potential transformed males have been tested, with no case of the desired type of fertility yet appearing. There were, however, occasional cases of spurious fertility due either to contamination or to lack of virginity somewhere in the sequence of matings, where it was essential to have virginity.

Tokunaga, Chiyoko. Notes on the sex chromosome constitution of oogonial cells in gynanders.

During the course of development a female zygote of D. melanogaster heterozygous for a ring X-chromosome may lose the ring chromosome from a cleavage nucleus, thus developing into a gynander. If the differentiation of germ cells should be the result not of their own genotype but of that of the somatic tissues of the gonads it is conceivable that XO oogonia could occur in the ovaries of gynanders.

175 gynanders were obtained among the progeny of a cross between y ac sn^3 females and the ring X-chromosome carrying X^{c2} f car males. In order to increase the frequency of gynanders among the progeny, y ac sn^3 virgin females were aged for eleven days at 17°C before mating. Of these 175 gynanders, one had underdeveloped gonads, eleven had one testis and one ovary, 43 had a pair of testes, and 120 had a pair of ovaries. The oogonia of 83 gynanders with 2 ovaries each were examined for their X-chromosomal constitution by means of the smear method, after fixation with aceto-lacto-orcein. 33 of these individuals clearly showed two X chromosomes, seven more seemed to have two X chromosomes, and the remaining 43 specimens did not show good mitotic metaphase figures. Oogonial mitotic figures of eight gynanders with one ovary and one testis were also investigated. One showed two X chromosomes clearly, while seven smears were unsuitable for cytological analysis.

Thus no mitotic figures were found which showed oogonial cells of XO constitution among the ovaries studied.

Wolff, M. and A. Coughlin. Tests for meiotic drive in interspecific hybrids.

The crosses recorded here were designed to detect possible cases of abnormal chromosome segregation in interspecific hybrids of D. pseudoobscura and D. persimilis. The rationale behind this test is to check on the possibility that during the divergency of these two species, there occurred in one or the other of the two a case of meiotic drive which became fixed and now would be undetectable by ordinary tests.

GENERAL PROCEDURE: Successive backcrosses with progeny counts each generation were carried out more or less extensively for a large number of cross types. For each type of cross, tests for possible abnormal chromosome segregation in the female and male were carried out in separate lines of backcrosses. For convenience, the following terminology was used: female Backcross -- female progeny with stock male
male Backcross -- male progeny with stock female

Mass matings were used in the original hybrid crosses. Pair matings were set up for each successive female Backcross. From the progeny of each generation of female Backcrosses, mass male Backcrosses were set up until successful crosses were obtained. Then a separate line of pair male Backcrosses were set up.

Where recessive markers were followed, + progeny were backcrossed to the original stock carrying the mutant. Where dominant markers were utilized, progeny showthe character were mated to the original wild type stock.

Although crossing-over was partially suppressed by normal or, for chromosome IV, special stock inversions, unequal recovery in the progeny of females might indicate unequal recovery of only the chromosomal region adjacent to the loci involved.

CHROMOSOME II:

Original Matings:

Iv. gl^2/gl^2 D. pseudoobscura females x wild type #12 D. persimilis males.
Iw. gl^2/gl^2 D. pseudoobscura females x wild type #21 D. persimilis males.
Iy. up Ba gl/up+gl D. pseud. females x wild type #21 D. persimilis males.
(Dominant marker followed)

Results:

Iv. 1st female Backcross -- Equal recovery - $\frac{gl^2/gl^{2+}}{283/\ 281}$

Iw. Equal recovery. Combined data through 3rd female Backcross - $\frac{gl^2/gl^{2+}}{456/\ 463}$

Iy. From 18 successful 1st female Backcross matings, 612 Ba and 924 Ba^+ progeny were obtained. However, practically all of the excess Ba^+ progeny came from only ten matings. Unfortunately, the female Backcross matings from these ten were singularly unsuccessful; the 2nd female Backcross yield were small and no successful 3rd female Backcrosses were obtained.

Matings from the remaining eight 1st female Backcross matings were more successful. These and subsequent backcrosses gave equal recovery in both sexes. Combined data for the 2nd through 6th female Backcrosses:

Ba		Ba+	
female	male	female	male
1160	829	1225	931

CHROMOSOME III:

Original Matings:

If. or (ST) D. pseudoobscura females x wild type #12 D. persimilis males
Ih. or (ST) D. pseudoobscura females x wild type #15 D. persimilis males
Ij. or (ST) D. pseudoobscura females x wild type #21 D. persimilis males

Results:

1st female Backcross: In all three there appeared to be an excess of or+ over or progeny:

	or	or+
If	1181	1453
Ih	508	636
Ij	463	686

Subsequent Backcrosses:

		or	or+
If. Equal recovery -	2nd B.C.	714	799
	3rd B.C.	2374	2413

	or	or+
Ih. Equal recovery 2nd Backcross -	167	164

No further matings.

Ij. While results of the 2nd female Backcross indicated possible preferential recovery of or+, subsequent backcross generations gave approximately equal recovery:

	or	or+
2nd B.C.	419	543
3rd B.C	618	691
4th B.C.	705	769

Backcrosses

If and Ij. Numerous successful male Backcrosses were obtained from 1st female Backcross progeny. In these and subsequent male Backcross generations there was approximately equal recovery of or and or+.

CHROMOSOME IV:

Original Matings:

Im. in hk j Cy (inv. IV)/1 D. pseudoobscura females x wild type #15 D. pers. males
Io. in hk j Cy (inv. IV)/1 D. pseudoobscura females x wild type #21 D. pers. males

Results:

	Cy	Cy+
Backcrosses: Im. Equal recovery 1st female Backcross:	228	245

Io. Equal recovery through 5th Backcross.

Combined data:

Cy	Cy+
3214	3316

male Backcrosses: Several successful backcrosses were obtained from the 3rd female Backcross progeny of Io. These and the 2nd male Backcrosses gave approximately equal numbers of Cy and Cy+ progeny. Combined data:

Cy	Cy+
419	364

X CHROMOSOME - RIGHT ARM:

Original Mating:

Wild type AH D. pseudoobscura males x se D. persimilis females

Result: 1st female Backcross - only one successful mating.

se	se+
61	59

No successful 2nd female Backcross matings.

X CHROMOSOME - LEFT ARM:

Original Matings:

IIf. y sn v co sh D. pseud. females x wild type #12 D. persimilis males
IIe. y xn v co sh D. pseud. females x wild type #21 D. persimilis males
It. Pt y sn v mbl D. pseud. females x wild type #21 D. persimilis males
(F_1 females mated to y sn v co sh D. pseudoobscura males)
Is. Pt y sn v mbl D. pseud. females x wild type #19 D. persimilis males
Dominant marker followed. (Pt males almost always y sn v)

Results:

IIf. 1st female Backcross only: Apparent excess of y^+ over y progeny:

y	y^+
209	324

Also an excess of sh^+ over sh progeny:

sh	sh^+
199	344

IIe. 1st Backcross only - Excess of y^+ over y progeny among both females and males.

y^+		y	
females	males	females	males
300	216	91	124
516		215	

Also a somewhat higher recovery of sh^+ over sh among the progeny.

sh^+		sh	
females	males	females	males
250	161	141	179
411		320	

It. Excess of + over y xn v progeny.

y sn v	+
331	707

Is. 1st female Backcross - Equal recovery:

Pt	Pt^+
307	282

Subsequent female Backcrosses: Equal recovery. Combined data 3rd through 6th female Backcrosses:

Pt	Pt^+
2059	2261

SEX RATION IN THE 3rd CHROMOSOME CROSSES:

(1) If. or (ST) D. pseud. females x wild type #12 D. persimilis males

Unfortunately no sex counts were made until the 3rd female Backcross.

3rd female Backcross:

Twenty-four matings were set up from nine 2nd female backcross matings. Overall, there was an approximately 2:1 ratio of females to males (3274 females to 1513 males) and an equal number of or^+ and or males (763 or^+ to 750 or males). Matings from two of the nine 2nd female Backcross matings seemed to show segregation for (a) high female excess and (b) only slight female excess. In the other lines, all matings gave off-ratios of approximately 2 females to 1 male or higher.

Subsequent Backcrosses:

Whenever only a slight excess of females appeared, all subsequent backcrosses descending from that mating gave similar sex ratios. When the ratio of females to males was high, the next backcross gave apparent segregation of approximately even and high female to male ratios. In some instances, or females from matings giving a high excess of females were mated to stock or D. pseudoobscura males. These also showed an apparent segregation for approximately even and high female to male ratios.

(2) Ij. or (ST) D. pseudoobscura females x wild type #21 D. persimilis males

This series consistently showed a relatively slight excess of females. The 3rd female Backcross gave 775 females to 699 males and counts from subsequent backcross generations were similar.

(3) Ih. Or (ST) D. pseudoobscura females x wild type #15 D. persimilis males

Data from only two 2nd backcross matings:

	females	males	males or	males or^+
Mating I	94	55	29	26
Mating II	147	35	16	19

Conclusion: There appears to be no evidence for any instance of meiotic drive incorporated into either of the two species.

Würgler, Friedrich E. Modification of x-ray induced embryonic mortality by different anoxia conditions before and during irradiation of uncleaved D. melanogaster eggs.

Continuing our work on the oxygen effect in Drosophila zygotes (see Ulrich & Würgler, DIS-33) a more detailed analysis of the influence of environmental gas conditions before and during irradiation has been made. In earlier experiments we have exposed eggs (which were 10 - 20 minutes old) to an air or a nitrogen current 1 minute before and during the 3 minutes lasting irradiation. In the experiments reported here the influence of prolonged pretreatment and change of gas atmosphere during irradiation has been tested; the age of eggs at the beginning of irradiation (3 minutes; 1000r; 50 kV; 10 mA) was again 10 - 20 minutes. A wild stock (Berlin wild) was used.

a) nitrogen treatment without irradiation
Unirradiated controls in air showed an embryonic mortality of 6.4%. 1 - 7 minutes nitrogen treatment increased the mortality linear with the duration to 8.7%. Beyond 8 minutes mortality increased more steeply. After 20 minutes in nitrogen 55.7% of the embryos died. Straight lines calculated by linear regression for these data were used to correct the following results (two different lines being calculated from (1) the interval 1 - 7 minutes and (2) the interval 8 - 20 minutes. In irradiation experiments anoxia was never extended over more than 8 minutes.
b) irradiation with nitrogen pretreatment
With 1 minute pretreatment and irradiation in nitrogen we found an embryonic mortality of 54.9% (n = 739). Longer (up to 5 minutes) and shorter (1/2 minute) pretreatment did not significantly change the results (summarized pretreatment experiments: 55.1%). The same was true even if there was no pretreatment at all: 54.7% mortality (n = 2488).
If the irradiation in air was preceded by exposure to nitrogen for 5 minutes, the mortality (84.1%; n = 1418) was not different from that without pretreatment (83.6%; n = 2426).
c) change of gas environment during irradiation
The rapid exchange of gas between egg and environment demonstrated under b) allowed for a change of the gas conditions within the egg during the 3 minutes lasting irradiation. In a first series X-rays were applied without an interval 1 minute (either the first, second, or third) in nitrogen and 2 minutes in air; in a second series 2 minutes in nitrogen and 1 minute in air. As compared to 3 minutes irradiation in air practically the same decrease of embryonic mortality was found in the 3 experiments of series 1. The same was true in series 2, where the decrease was more pronounced. Therefore the results of each series are summarized. Thus an embryonic mortality of 77.2% (1 minute N_2) and 69.2% (2 minutes N_2) was found. This agrees well with the expectation of 77.2% and 68.0%. This expectation is derived from the hypothesis of independent realization of radiation-damage caused under aerobic and anaerobic conditions. The calculation of the exact data were based on the findings that dose-action curves in air (ULRICH 1960) and nitrogen (WÜRGLER 1960) can be approximated by curves of the form $y = 1 - e^{-kd}$ (y - mortality; D = dose).
d) conclusions
1.) The replacement of gas inside D.m. zygotes by N_2 is achieved within a few seconds.
2.) A nitrogen pretreatment up to 5 minutes has no influence on the embryonic mortality induced by irradiation of 10-20 minutes D.m. eggs.
3.) If during part of the irradiation time the air atmosphere was replaced by nitrogen, the embryonic mortality decreased proportionately to the length of the N_2-fraction. This agrees with the assumption of independent realization of the radiation-damage caused under aerobic and anaerobic conditions.

(Work supported by Schweiz. Nationalfonds zur Förderung der wissenschaftlichen Forschung.)

Zimmering, S. and H. J. Muller.

Studies on the action of the dominant female-lethal Fl and of a seemingly less extreme allele, Fl^s.

Tests were made to determine whether the female lethal Fl (Muller and Zimmering, 1960, Genetics 45: 1001-1002) still acts as a complete lethal when present in pseudo-males having two Fl-containing X-chromosomes and two third chromosomes containing tra ("transformer of sex," Sturtevant, 1945, Genetics 30: 297). It was found that the sex transformation failed to save the lives of these flies. Similarly, in flies heterozygous for Fl, the viability of pseudo-males (XX, but homozygous for tra) was as much reduced by the dominant action of Fl as was the viability of their non-transformed sisters that had the same X-chromosome composition but were heterozygous for tra.

Tests of the genetic factors determining the dominant female-lethal effect of Fl in crosses of our y v stock ("b120") have shown that all the major chromosomes (X, II, and III) of this stock play an important and synergistic role in producing the effect. In daughters of females heterozygous for some or all of of these three chromosomes little or no dominant lethal effect was produced except when all three of the chromosomes were present together in the given mothers. The effect was a maternal

one, tending to kill the heterozygous Fl (but not the non-Fl) daughters of all classes, provided that the mothers contained these intensifiers in all three chromosomes, in at least single dose. The intensifiers themselves were partially dominant, in that mothers homozygous for them gave a higher lethality of daughters heterozygous for Fl than did mothers heterozygous for them. When virgin females carrying the intensifiers are kept at a comparatively high temperature (35°C) for 36 or more hours prior to egg laying, the mortality of their heterozygous Fl daughters, derived from eggs laid at 25°C within the next three days, relative to that of their brothers, is considerably reduced (in the cases studied, from about 96% to 80%). Little reduction of mortality is produced when the exposure to warmth is allowed to last only 24 hours. Other cases of genes that have a maternal effect in killing daughters but not sons have been reported by Redfield (1924, 1926), Gowen and Nelson (1942), Gowen (1949), and Bell (1954), but in these cases there was no finding of a primary female-lethal gene, corresponding to Fl, that had to be present in the female that was herself subject to the lethal action.

In the experiment on pseudo-males the stock that had been used to provide the tra gene (our stock "j22") had had females with attached X's and males whose single X-chromosome contained w^a. The crosses of this stock unexpectedly showed that the w^a-containing chromosome also carried an allele of Fl. We are, for reasons to be given below, denoting this as Fl^s (a symbol superseding our earlier, unpublished designation, Fl^2). It was found that compound females, one of whose X-chromosomes carried Fl and the other Fl^s, invariably died. However, when crosses were made of Fl^s males to stocks y v ("b120") and w ("b69"), which on crossing to Fl males had given a high mortality of daughters, i.e., a high dominance of Fl, no such lethality occurred among these daughters. That is, Fl^s, unlike Fl, failed to act as a (partially) dominant lethal. That this difference was not sufficiently explained by autosomal modifiers was proved by experiments in which the autosomes were appropriately substituted by the aid of chromosomes having inversions and markers. Similarly, parts of the X far from Fl were ruled out. It was further found that Fl^s, unlike Fl, is not, when in its original setting, lethal even in the female homozygous for it, despite its lethality when "in compound" with Fl. Fl^s does sometimes act as a lethal to females homozygous for it, however, when taken out by crossing over from its original genetic setting, but the number and loci of the modifying genes here involved have not been worked out.

In the crosses that gave rise to homozygous Fl^s females it was found that these females are invariably sterile (hence the superscript s). Their abdomens remain unenlarged, like those of homozygous fes females, while they appear normal in other outward respects. Like the lethality of Fl, the sterility of Fl^s is to a certain extent and under some conditions dominant, inasmuch as heterozygous Fl^s females are found in some crosses to have a high frequency of sterility. Such sterility has not thus far been observed among heterozygous Fl females. Further studies are however needed to determine definitely whether there are differences in the action of Fl and Fl^s when they are in exactly the same genetic setting.

Whereas Fl arose within Inversion-49, Fl^s is in an X-chromosome of normal structure. It must therefore have arisen as a result of a spontaneous mutation independent of that which produced Fl. Both these genes have been found by linkage tests to be slightly to the left of oc. Fl^s has been located more exactly as lying between cm and ct, nearer to cm, inasmuch as only 2 out of 10 tested crossovers between cm and ct^6 proved to have been between the loci of cm and Fl^2, while the rest were between the loci of Fl^2 and ct, thus placing Fl^s at approximately 19.1 in the X-chromosome map.

Thus far, tests for female lethality have been made in our laboratory, by Robert Baum and by Marcia Henning, of a considerable number of our stocks in which the X-chromosome of the male had been kept confined to males by having them always crossed to females with attached X's. Thus far, no cases of female lethality have been found other than those in which the original Fl allele had been present as a result of the common origin of the Fl-containing region of the given X-chromosome with that of the X-chromosome of the stock in which Fl had first been discovered. These results suggest that mutations of the type in question are comparatively rare.

(Work supported by a grant from the U.S. Public Health Service (Contract RG-5286(C3)).)

Browning, Luolin S. and Edgar Altenburg. Weighing of dehydrated Drosophila as a counting method.

The counting of many thousands of individual flies is one of the major technical problems involved in experiments in which there is a low incidence of the phenomenon being studied -- for example, visible mutation rates at specific loci and pseudoallelic crossingover. A method which eliminates this task without the use of special equipment and furnishes counts reliable within a standard error of about 3% has been developed.

Flies are grown by the "vat method" (D.I.S. #33, p. 177), from 8,000 to 20,000 offspring being obtained from several transfers of the same parents (usually 200 to 400 parents). Since the flies that hatch first in a vat may be twice as large as those hatching later, due largely to better cultural conditions, it was found necessary to establish a rigid routine for emptying, examining, and handling the flies. Three examinations of the etherized offspring are made per vat -- on the 14th, 17th and 20th days -- after which the vat is discarded. After each examination, the offspring are immediately killed with ether, placed in an open milk bottle in an incubator at 55°C and kept for at least 24 hours. All the flies from a given vat are eventually added to the bottle, and after dehydrating, the bottle is transferred to a dehumidifier until a convenient time for weighing on an analytical balance. Flies kept for as long as three months in the dehumidifier showed only a slight change in weight. It was found that various mutant strains in laboratory use had different weights and sex ratios, and although this variation was not great, it was calculated by actual counts for different experiments. The table below shows the results of such a sampling, together with the weights after dehydration of each sample of counted flies coming from a single vat. (The female parent flies in this experiment carried w^a in one X chromosome and w^{Bwx} in the other, and were also heterozygous for y and spl, as well as for the Cy and Ubx inversions in the second and third pair of autosomes; the male parents were Basc males containing sc w^a B.)

Females			Males			Females and Males		
No.	Weight (Grams)	No. per Gram	No.	Weight (Grams)	No. per Gram	No.	Weight (Grams)	No. per Gram
4,637	1.2920	3,560	3,903	0.8543	4,580	8,540	2.1463	3,980
2,455	0.7533	3,260	2,184	0.4851	4,500	4,639	1.2384	3,750
1,973	0.8127	2,436	1,563	0.4139	3,780	3,536	1.2266	2,880
-	-	-	-	-	-	2,797	1.0342	2,700
-	-	-	-	-	-	2,551	0.7686	3,320
-	-	-	-	-	-	2,241	0.7296	3,080
2,072	0.6431	3,220	1,724	0.3969	4,320	3,796	1.0400	3,650
-	-	-	-	-	-	2,832	0.7465	3,790
2,406	0.7313	3,290	2,091	0.4749	4,420	4,497	1.2062	3,730
-	-	-	-	-	-	5,375	1.6378	3,280
-	-	-	-	-	-	4,141	1.2296	3,375
-	-	-	-	-	-	5,657	1.6760	3,380
13,543	4.2324	3,200	11,465	2.6251	4,370	50,602	14.6798	3,450 ± 113(S.E.)

There is a range in yields among the twelve sample vats of from 2,200 to 8,500 flies, with a range in flies per gram of from 2,700 to 3,980. The average number of flies per gram for all the vats is 3,450±113, or 3,450±3.3%. Thus in 67% of all experiments involving 50,000 or more flies, the total number of flies (obtained by multiplying the weight of dehydrated flies by 3,450) would be expected to be in error by no more than 3.3%, and by no more than 6.6% in 95% of such experiments.

It is sometimes desirable to scan rapidly a large number of flies for rare mutants in one sex but without separating them as to sex. The table shows that the number of females represented by one gram of dehydrated males and females can be reliably estimated. For example, the sex ratio based upon a count of about 25,000 flies (columns one and four in the table) was 0.54 females: 0.46 males, 13,543 females being included in a total weight of 7.3508 grams of both males and females. This indicates that one gram of both males and females contained 1,845 females, a figure very similar to that of 1,863 females obtained when the average number of males

and females contained in one gram of both males and females (3,450) based upon a count of over 50,000 flies is multiplied by the sex ratio factor of 0.54.

Mickey, G.H. Nigrosine as an aid for staining brain and salivary gland chromosomes.

We have found that the addition of nigrosine (alcohol soluble -- National Aniline) to aceto-orcein stain enhances the staining of chromosomes remarkably. Larval ganglion chromosomes are stained intensely enough to be examined and photographed without the use of phase contrast microscopy. The procedures are as follows:

1) Aceto-orcein stain is prepared by dissolving one gram of orcein dye (Allied Chemical) in 100 ml of 45% acetic acid in an Erlenmeyer flask, with reflux condenser attached, and heating for one hour without boiling. The solution is cooled, filtered, and stored in refrigerator.

2) Aceto-nigrosine stain is prepared by heating 100 ml of 50% acetic acid to boiling point, adding 4 gms of alcohol soluble nigrosine and stirring constantly. It should be boiled for 3 - 5 minutes, or until it acquires a highly viscous consistency. When cooled, it should be filtered, using a water pump filter. Store in closed glass vessel in the dark at room temperature.

3) Aceto-orcein-nigrosine stain for salivary slides is made by mixing 1 ml of aceto-nigrosine and 9 ml of aceto-orcein stain. This must be filtered before each use!

4) Aceto-orcein-nigrosine stain for brain slides is made by mixing 4 ml of aceto-nigrosine and 6 ml of aceto-orcein stain. Must be filtered before each use!

The chief drawback of the stain is the tendency for the nigrosine to precipitate, which necessitates frequent filtering. A temporary squash preparation sealed with cover glass wax will improve with aging a few hours to several days, but should be made permanent if it is to be kept for a longer time. We use the dry ice technique for removing the cover glass and mount in euparal. The stain is permanent and does not fade.

The stain produces superb salivary gland slides, the fine bands on the chromosomes showing very distinctly.

Moyer, S. E., R. E. Comstock and L. H. Baker. Efficient procedures for culturing Drosophila in disposable paper containers.

Arnold (Amer. Biology Teacher 19:248-251 and DIS-32 p. 166) described a method for utilizing plastic-lined paper containers for culturing Drosophila melanogaster. We have modified this basic plan to enable greater ease and speed in routine handling of large numbers of containers.

Covers for the containers are now available with a half moon clear plastic window. Ventilation holes, if needed, can be quickly punched with a "pin cushion" made from a rubber laboratory stopper and well spaced common pins.

The medium is anchored in the bottom of the container by a 4" x 4" non-sterilized 8 ply surgical gauze sponge stapled to the sides. This prevents the medium from falling on the flies while the container is inverted during anesthesia.

Anesthesia is accomplished by applying several drops of ether on a small cotton plug stapled into a 1/4 inch hole punched in the side of the container. Since this plug may be an attraction as a site for pupation, it may be desirable to locate it near the top of the container. Further diversion of the larvae from the plug may be achieved by limiting the amount of medium to slightly more than needed to cover the gauze on the bottom. In this way, larvae are attracted to the remaining gauze above the level of the medium. However, ether does not kill pupae occasionally lodged in plugs and possible physiological effects seem negligible.

Although anesthesia is slow with this system, waiting can be eliminated by initiating anesthetization well in advance of anticipated use of a particular culture. There is no danger of over-etherization to flies remaining in containers for an extended period of time prior to examination.

The chief advantage of using these paper containers is in the time and money saved. The cost of the unit and the time required for assembling it by the above procedures is considerably less than the time and cost of labor for washing and sterilizing bottles. Also, each of these containers supports a larger population for a longer period of time than does a half pint bottle. Finally, the containers can be stacked on top of each other for maximum utilization of storage space for Drosophila cultures.

von Borstel, R. C. and Margaret M. Fine. A medium suitable for hatchability and eclosion tests.

Since charcoal agar is a nuisance to mix and since it results in reduced viability of Drosophila larvae, another agent was sought that would give satisfactory contrast to the Drosophila culture medium for hatchability testing.

The obvious choice was fruit juice and it was found that frozen grape juice concentrate works well. The flies cling to the medium with a tenacity that decries the change of name from ampelophila. Egg production and survival are high, and eclosion tests as well as hatchability tests are possible from the same vial.

The food formula used at Oak Ridge corresponds closely to that used at the California Institute of Technology (Lewis, DIS-34, 117, 1960). To prepare the hatchability medium, one 12-ounce can of frozen grape juice is stirred into every two liters of the Drosophila food and the mixture is then poured into vials. This food is just firm enough for egg hatchability testing, and it may be desirable to add more agar if some other food formula is used.

We presume that other fruit juice concentrates could be used as well, and appropriately dark wines could possibly be used, but local regulations are such that experimentation along these lines at Oak Ridge has been severely restricted.

PERSONAL AND LABORATORY NEWS

James Divelbiss is joining the staff o the Biology Department, Westmar College, Le Mars, Iowa in September 1961, and plans to establish a Drosophila research laboratory. Since library facilities in the area of genetics are limited he would appreciate receiving any available reprints, new or old.

Edward C. Keller, Jr., has moved from The Pennsylvania State University to The University of North Carolina at Chapel Hill in The School of Medicine, Department of Biochemistry and Nutrition.

Maxi E. Krawinkel, curator of stocks and head technician of the Purdue University Drosophila laboratory for the past five years, was killed in an automobile accident while on vacation in Michigan on January 22, 1961. She has been buried near her home in Bern, Switzerland. A memorial book collection bearing her name has been established in the Biology Library, Purdue University.

MATERIALS REQUESTED OR AVAILABLE

The inbred temperature lines of D. melanogaster derived from a wild population near State College, Pennsylvania, that are described in the stock list of University Park, Pennsylvania (DIS-34) will be maintained until further notice is given in D.I.S.

James Divelbiss is currently engaged in a pseudoallelic investigation of the brown locus. He would appreciate receiving stocks of any brown alleles except the following which he already has: bw^{1}, bw^{81}, bw^{75}, bw^{59}, bw^{M58}, bw^{Mi59} and bw^{Am}. For his address see entry in Personal and Laboratory News.

ANNOUNCEMENTS

Drosophila melanogaster Stock Centers

The National Science Foundation is now supporting two stock centers for the maintenance of strains of Drosophila melanogaster. One is located at the California Institute of Technology, Pasadena, California and is under the direction of Professor E. B. Lewis while the other one is located in the Division of Chemotherapy of The Institute for Cancer Research, Philadelphia 11, Pennsylvania and is under the direction of Dr. I. I. Oster. The nucleus of both centers will consist of duplicates of the 800 basic stocks hitherto only maintained in Pasadena. These centers will serve

as a source of supply for virtually any research needs which might arise regardless of field of genetic interest, 2) provide insurance against loss of all or part of their respective collections by some unforeseen catastrophe, 3) allow the centers to make replacements of each other's stocks in the event any have broken down and 4) provide the "marker" and tester stocks and other combinations of mutations useful in research and teaching.

In addition to the 800 basic stocks, the Center in Philadelphia will maintain approximately 1600 other strains. These will include 600 stocks representing the major portion of the strains currently maintained by Professor H. J. Muller at Indiana University, 200 strains which had been maintained by Dr. J. Schultz in the Department of Genetics of The Institute for Cancer Research, and 800 strains consisting of other useful mutations, combinations of them, and multiple alleles of loci of unusual interest obtained from other laboratories or synthesized by the Center.

We would like to suggest that other workers should contribute useful stocks for inclusion in the collections. The main requisites for acceptance of such strains are that they be held in a combination not requiring selection, they represent new loci, alleles of unusual interest, improved balancers, etc., and they be free of mites. Stocks of mutants which overlap wild-type or of biochemical mutants which have no morphological effect should contain an RK1 marker mutant to serve as a check on the possibility of contamination. In order to avoid the inclusion of too many stocks of questionable usefulness in the permanent collection, consultations with the Subcommittee on Drosophila Stocks will be held from time to time concerning the advisability of adding any of these newly contributed stocks or of eliminating old ones. Those who wish to contribute stocks should send a complete description of the stock to the Center ahead of time preferably on 3 x 5 cards. This will facilitate evaluation of the stock and provide the basic information needed for the stock records.

When a stock has been improved (for example, by introducing a more efficient balancer, or a more useful combination of mutants) the old stock in general will be discarded and replaced by the new one. However, no mutant type, balancer, nor any chromosomal rearrangement will be deliberately discontinued without notice being given in the Drosophila Information Service at least one year in advance.

Requests for stocks for research purposes will be filled as promptly as possible. As heretofore, there will be no charge for this service. It is requested that the receiver return the empty plastic vials in the original mailing carton.

Suggestions for improving the stockkeeping service are welcomed and can be addressed to either Center or to the Subcommittee on Drosophila Stocks.